STUDY ON ETIOLOGY AND CLINICAL PROFILE OF DILATED CARDIOMYOPATHY

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CERTIFICATE

This is to certify that the dissertation entitled "**STUDY ON ETIOLOGY AND CLINICAL PROFILE OF DILATED CARDIOMYOPATHY**" is a bonafide work done by **Dr.C.R.SRINIVASAN**, at Madras Medical College, Chennai in partial fulfilment of the university rules and regulations for award of M.D., Degree in General Medicine (Branch-I) under my guidance and supervision during the academic year 2009 - 2012.

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I solemnly declare that this dissertation entitled "**STUDY ON ETIOLOGY AND CLINICAL PROFILE OF DILATED CARDIOMYOPATHY"** was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, during 2009-2012 under the guidance and supervision of **Prof.E.DHANDAPANI, M.D.** This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree in General Medicine (Branch-I).

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LIST OF ABBREVIATIONS

DCM	DILATED CARDIOMYOPATHY
HF	HEART FAILURE
CCF	CONGESTIVE CARDIAC FAILURE
ECHO	ECHOCARDIOGRAM
ECG	ELECTROCARDIOGRAM
PPCM	PERIPARTUM CARDIOMYOPATHY
LV	LEFT VENTRICLE
RV	RIGHT VENTRICLE
WHO	WORLD HEALTH ORGANISATION
AHA	AMERICAN HEART ASSOCIATION
LVNC	LEFT VENTRICULAR NON COMPACTION
ARVD	ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA
BNP	BRAIN NATRIURITIC PEPTIDE
AF	ATRIL FIBRILLATION
RBBB	RIGHT BUNDLE BRANCH BLOCK
LBBB	LEFT BUNDLE BRANCH BLOCK

CONTENTS

INTRODUCTION

Cardiomyopathies are an important and heterogeneous group of diseases for which an understanding in both the public and medical community has historically been impaired by confusion surrounding definitions and nomenclature. Classification schemes, of which there have been many, are useful in defining and drawing relationships or distinctions between these complex diseases for the purpose of promoting greater clarity.

Inadult population the prevalence of heart failure is estimated to be about 1 to 1.5%. The mortality and morbidity remain high (median survival of 3.2 years for women and 1.7 years for men).Up to 25% of all cases of CHF is caused byDilatedcardiomyopathy.The incidence and prevalence of CHF due to cardiomyopathy appears to be increasing. The incidence of DCM is reported to be 5 to 8 cases per 1,00,000 population per year. When compared to females DCM is 3 times more common in males. The frequency of occurrence is also more common in blacks[1].

Many classifications in the literature are to some degree contradictory in design, and indeed none of the proposed schemes can be regarded as ideal. The dilemma is caused by the heterogeneity in the presentation of this diverse group of diseases. A previous prominent classification of cardiomyopathies (1995) was represented in a very brief document under the auspices of the World Health Organization (WHO) [2].

However, with the identification of new diseases over the past decade, and dramatic advances in cardiovascular diagnosis and knowledge regarding etiology, some disease definitions have become outdated and the WHO classification rendered essentially obsolete. Indeed, the past several years has witnessed a rapid evolution in the molecular genetics of cardiology. In particular, ion-channelopathies have emerged as conditions predisposing to potentially lethal ventricular tachyarrhythmias, caused by mutations in proteins leading to dysfunctional sodium, potassium, calcium, and other ion channels.

Recently, under the auspices of the American Heart Association, a contemporary classification of cardiomyopathies has been presented, relying substantially on recent advances in the characterization of diseases affecting the myocardium. The new classification scheme affords a large measure of clarity to this area of investigation and facilitates interaction among the clinical and research communities in assessing the diagnosis, prognosis, and management of these complex diseases.

The natural history of DCM remains incompletely understood. This is because this diagnosis clearly contains a variety of causes and patients have highly variable presentations. The presentations of patients can range from asymptomatic left ventricular dysfunction to mild, moderate, or severe congestive heart failure. Different studies report wide-ranging

estimates of annual mortality that are between 10% and 50%. Traditionally, it is held that symptomatic heart failure is invariably progressive. However, several factors suggest that this concept should be re-examined and that biologic factors may determine favourable or unfavourable long-term outcomes.

The prognosis of DCM may be much more variable than previously appreciated.Several features of the clinical presentation may be valuable in predicting patient outcome [3]. In addition, the underlying etiology of the cardiomyopathy clearly has a substantial impact on the natural history, thus warranting an exhaustive search for causes. Some cardiomyopathies have excellent long-term survival, whereas others, particularly amyloidosis and human immunodeficiency virus (HIV)–related disease, carry grave prognoses.

With theadvancement in molecular genetics and identification of underlying etiologies, dilated cardiomyopathy is being mentioned as a specific diagnosis and not by exclusion. The most common indication for cardiac transplantation in west is DCM.[4,5]

In view of high prevalence of heart failure and also lack of data on dilated cardiomyopathy this study was undertaken. The ECG and echocardiography were also evaluated in the present study.

AIMS & OBJECTIVES

Aim of the study is:

1. To evaluate 100 cases of dilated cardiomyopathy and identify various etiology factors.

2.To analyze the clinical profile of patients with dilated cardiomyopathy

3.To study the electrocardiographic and echocardiographic profile of these patients

REVIEW OF LITERATURE

Historical aspects

The term cardiomyopathy was first introduced in the year 1957 by Wallace Brigden of the National Heart Hospital, London to refer collectively a primary myocardial disease. Congenital, rheumatic, hypertensive or coronary artery disease were excludedby strict definition of myocardial hypertrophy, dilatation.To differentiate between ischemic and non-ischemic cardiomyopathies, Felker and associates used angiographic studies.

In 1970 Burch and his associates used the term ischemic cardiomyopathy when they found that coronary artery disease can ultimately lead to myocardial dysfunction which is out of proportion to the level of ischemia or infarction. He also found that early revascularization can prevent the development of ischemic cardiomyopathy.

The Framingham Heart Study showed that the heart failure is found to be twice in diabetic men and also five times more in diabetic women when compared with age-matched control subjects. This increased incidence of heart failure in diabetic patients persisted despite correction for age, obesity, hypercholesterolemia, hypertension and coronary artery disease.

It was Rubler et al who first identified the existence of a diabetic cardiomyopathy in patients who had no evidence of coronary atherosclerosiswith congestive heartfailure. Chronic alcoholism can lead to cardiomyopathy, which was first described by Rubin et al. He also found that alcohol is toxic to cardiac muscle in a dose dependent manner.

A syndrome of idiopathic heart failure in the early post partum period was first described by Ritchie et al. This was first described as post-partum cardiomyopathy and later replaced by peripartum cardiomyopathy. Majority of case presents during early post partum period but certain number cases can present even during last months of pregnancy. The criteria for diagnosis of peripartum cardiomyopathy were first described by Demakis et al.

Echocardiographic and macroscopic features of dilated cardiomyopathy in HIV were first described in 1986 by Cohen and colleagues, which later came to be known as HIV cardiomyopathy.

Cardiomyopathies were classified into three categories namely congestive, hypertrophic and restrictive by Goodwin et al in 1970s .This classification is widely used and still valid in many circumstances. The term cardiomyopathy was to be used only for myocardial disease of unknown cause, proposed by WHO in 1980. In 1995, the WHO committee suggested a new classification system for dilated cardiomyopathy that includes a wide variety of myocardial diseases.

Recently, under the auspices of the American Heart Association (2006), a contemporary classification of cardiomyopathies has been presented,[15] relying substantially on recent advances in the characterization of diseases affecting the myocardium.[16-18] The new classification scheme affords a large measure of clarity to this area of investigation and facilitates interaction among the clinical and research communities in assessing the diagnosis, prognosis, and management of these complex diseases. This classification takes the place of the WHO document, but as new data emerge it also will undoubtedly require further review and revision.

Definitions

The proposed definition of cardiomyopathies is: *A heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction, which usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilatation, and are due to a variety of etiologies that frequently are genetic. Cardiomyopathies are either confined to the heart or are part of generalized systemic disorders, often leading to cardiovascular death or progressive heart failure-related disability.*

Classification

Most cases of heart failure are caused by heart muscle disease (cardiomyopathy). Within the classification of cardiomyopathies [6,7], the

most common cause of the clinical syndrome of heart failure is a *secondary* (ischemic, valvular, hypertensive, and so on) or a *primary* (genetic, nongenetic, acquired) *DCM*, defined as a ventricular chamber exhibiting increased diastolic and systolic volumes and a low (<45%) ejection fraction [8]. The natural history of the clinical syndrome of heart failure depends on the course of myocardial failure because (1) the most powerful single predictor of outcome is the degree of left ventricular (LV) dysfunction as assessed by the LV ejection fraction [9] (2) treatment that improves intrinsic ventricular function improves the natural history of heart failure; and (3) treatment that ultimately worsens intrinsic function, such as many types of positive inotropic agents, is associated with an adverse effect on outcome [10].

The 1995 World Health Organization/International Society and Federation of Cardiology (WHO / ISFC) classification of cardiomyopathies[7] was recently revised to accommodate several rapidly emerging realities, particularly the identification of new disease entities, advances in diagnosis, and knowledge of etiology of previously unknown types of heart muscle disease.[6]

The new classification of cardiomyopathies is described in the WHO/ISFC classification of cardiomyopathy was mainly based on the global anatomic description of chamber dimensions in diastole and systole. Thus, therestrictive and dilated categories had definitions based

on left ventricular dimensions or volume, which also define function via calculated ejection fraction. The justification for this is that these two groups have distinct natural histories and respond differently to medical treatment. The novel AHA Scientific Statement emphasizes the genetic determinants of cardiomyopathies.

Thus, dilated and restrictive cardiomyopathies are defined as *mixed* cardiomyopathies (predominantly nongenetic); however, hypertrophic cardiomyopathy (HCM), which is caused by mutations in contractile proteins, and other rare forms of cardiomyopathy, including arrhythmogenic right ventricular (RV) cardiomyopathy/dysplasia (ARVC/D) and LV noncompaction (LVNC), which also turned out to be completely genetic in basis, are defined *genetic* cardiomyopathies. The third category concerns *acquired* cardiomyopathies, such as peripartum and tachycardia-induced cardiomyopathies. Conversely, genetic cardiomyopathies without unique phenotypes and involvement of a generalized multiorgan disorder, such as the DCM of Becker-Duchenne, are defined as *secondary* cardiomyopathies. This distinction is arbitrary and may inevitably cause significant overlap between primary and secondary cardiomyopathies.

Classification of the causes of cardiomyopathy continues to be a challenge, and a satisfactory and uniformly agreed on classification system remains in evolution[11].Classification schemes are plagued by the

fact that as the causal basis of heart muscle disease becomes increasingly understood, it is also appreciated that for a given etiology, there may be a spectrum of phenotypes that can overlap or evolve. Recently, a new classification of cardiomyopathies that incorporates molecular insights was proposed by an American Heart Association Scientific Statement panel.[11]

This classification divides cardiomyopathy into primary and secondary causes, in a manner similar to traditional classification schemes, but adds important sub characterization of the primary cardiomyopathies into genetic, mixed, and acquired groups. From the clinical perspective, where the objective is diagnosis and delivery of effective therapy that may be cause-specific, there is major overlap with the concept of an acquired primary cardiomyopathy and a secondary cardiomyopathy. An important new addition to the genetic subgroup is that of ion channel disorders, which often are not accompanied by structural heart disease but clearly can be considered a primary disorder of the heart.

Based on all these considerations, the recommendation is that cardiomyopathies can be most effectively classified as

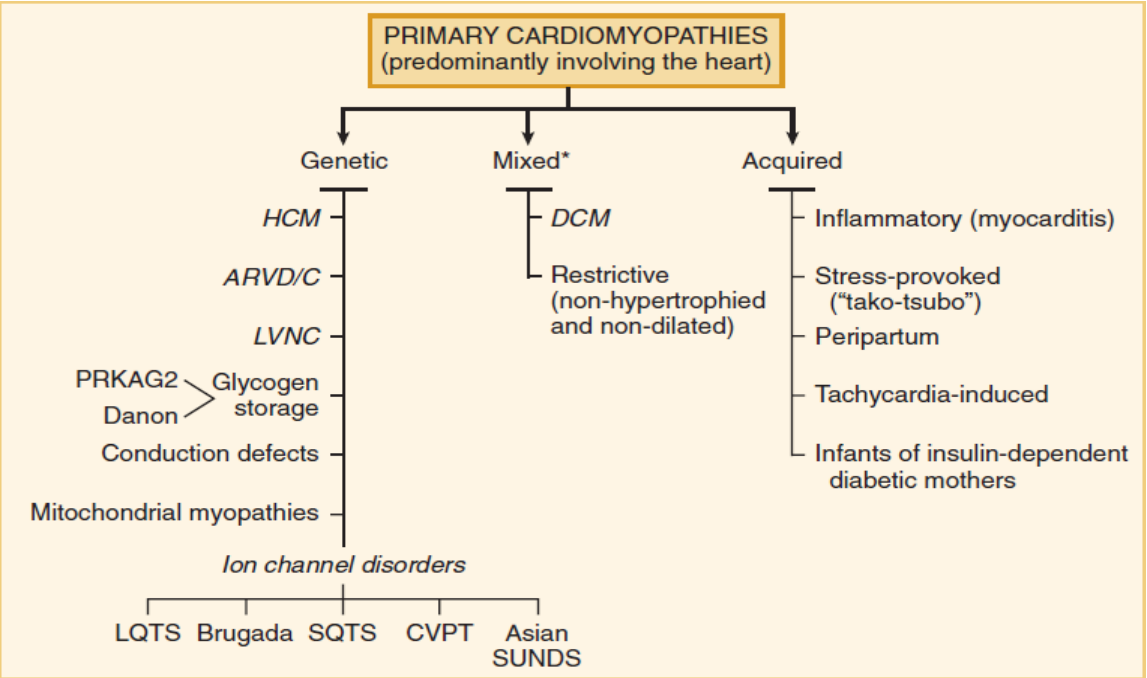
Primary-genetic
Primary-mixed (i.e., genetic and nongenetic)
Primary-acquired
Secondary

CLASSIFICATION OF CARDIOMYOPATHIES

Dilated cardiomyopathy	Dilation and impaired contraction of the left ventricle or both ventricles Caused by familial-genetic, viral, immune, alcoholic-toxic, or unknown factors or is associated with recognized cardiovascular disease
Hypertrophic cardiomyopathy	Left and/or right ventricular hypertrophy, often asymmetric, which usually involves the interventricular septum Mutations in sarcoplasmic proteins cause the disease in many patients
Restrictive cardiomyopathy	Restricted filling and reduced diastolic size of either ventricle or both ventricles with normal or near-normal systolic function Idiopathic or associated with other disease (e.g., amyloidosis, endomyocardial disease)
Arrhythmogenic right ventricular cardiomyopathy	Progressive fibro fatty replacement of the right, and to some degree the left, ventricular myocardium Familial disease is common
Unclassified cardiomyopathy	Diseases that do not fit readily into any category; examples include systolic dysfunction with minimal dilation, mitochondrial disease, and fibroelastosis
Specific Cardiomyopathies	
Ischemic cardiomyopathy	Arises as dilated cardiomyopathy with depressed ventricular function not explained by the extent of coronary artery obstructions or ischemic damage
Valvular cardiomyopathy	Arises as ventricular dysfunction that is out of proportion to the abnormal loading conditions produced by the valvular stenosis and/or regurgitation
Hypertensive cardiomyopathy	Arises with left ventricular hypertrophy with features of cardiac failure related to systolic or diastolic dysfunction
Inflammatory cardiomyopathy	Cardiac dysfunction as a consequence of myocarditis
Metabolic cardiomyopathy	Includes a wide variety of causes, including endocrine abnormalities, glycogen storage disease, deficiencies (such as hypokalemia), and nutritional disorders
General systemic	Includes connective tissue disorders and infiltrative diseases

disease	such as sarcoidosis and leukemia
Muscular dystrophies	Includes Duchenne, Becker-type, and myotonic dystrophies
Neuromuscular disorders	Includes Friedreich ataxia, Noonan syndrome, and lentiginosis
Sensitivity and toxic reactions	Includes reactions to alcohol, catecholamines, anthracyclines, irradiation, and others
Peripartum cardiomyopathy	First becomes manifested in the peripartum period, but it is probably a heterogeneous group

PRIMARY CARDIOMYOPATHIES



Source: AHA Scientific statement from the council on clinical cardiology, Circulation 113:1807, 2006

Dilated Cardiomyopathy

The hallmarks of dilated cardiomyopathy (DCM), the most common cardiomyopathy, are enlargement of one or both of the ventricles and systolic dysfunction. It is not uncommon for chamber enlargement to precede signs and symptoms of congestive heart failure. Recent classification revision attempts recognize that chamber dilation is

part of the spectrum of genetic and environmental disorders affecting the heart; thus, a patient presenting with DCM may have a broad array of cardiac or systemic conditions. Nevertheless, DCM is an important and frequent clinical presentation. In 50% or more of patients with a DCM, an etiologic basis will not be identified, in which case the patient is referred to as having an idiopathic DCM.[12,13]

Natural History

The natural history of DCM remains incompletely understood. This is because this diagnosis clearly contains a variety of causes and patients have highly variable presentations. The presentations of patients can range from asymptomatic left ventricular dysfunction to mild, moderate, or severe congestive heart failure. Different studies report wide-ranging estimates of annual mortality that are between 10% and 50% [14]. Traditionally, it is held that symptomatic heart failure is invariably progressive. However, several factors suggest that this concept should be re-examined and that biologic factors may determine favourable or unfavourable long-term outcomes.[15]

First, there has been an impact of therapy on the natural history of patients. Whereas the 1-year mortality in the placebo arm was approximately 50% in the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS) conducted in the 1980s, similar patients experienced ~20% annual mortality in the Carvedilol Prospective

Randomized Cumulative Survival (COPERNICUS) trial conducted in the 1990s, and this dropped further in the 2000s to ~10%. There is also growing awareness that treatment with pharmacologic therapies that antagonize the neurohormonal system can lead to myocardial recovery or "reverse left ventricular remodelling" in some patients with DCM. Finally, it is reported that between 25% and 33% of patients presenting with new-onset DCM experience meaningful cardiac recovery.[16]

Prognosis

The prognosis of DCM may be much more variable than previously appreciated.[14,15]Several features of the clinical presentation may be valuable in predicting patient outcome. In addition, the underlying etiology of the cardiomyopathy clearly has a substantial impact on the natural history, thus warranting an exhaustive search for causes. Some cardiomyopathies have excellent long-term survival, whereas others, particularly amyloidosis and human immunodeficiency virus (HIV)–related disease, carry grave prognoses.[13]

A study using microarray analysis to measure gene expression in endomyocardial tissue obtained from patients suggested that patients who have favourable long-term outcomes accompanied by reverse remodelling can be detected at the time of clinical presentation.[15] Alternatively, it is clear that some patients may experience sudden deterioration after a period of stability or never experience a quiescent

time.[17] It is also critical to appreciate that certain patients may have severe and life-threatening hemodynamic embarrassment at initial presentation. For these patients, a diagnostic evaluation including endomyocardial biopsy should be rapidly performed; these patients are critically ill and frequently require inotropic or mechanical support as a lifesaving therapy.[18]

The determinants of the natural history are not entirely clear, but several studies suggest that biomarkers or panels of laboratory values may have prognostic value.[14,15]

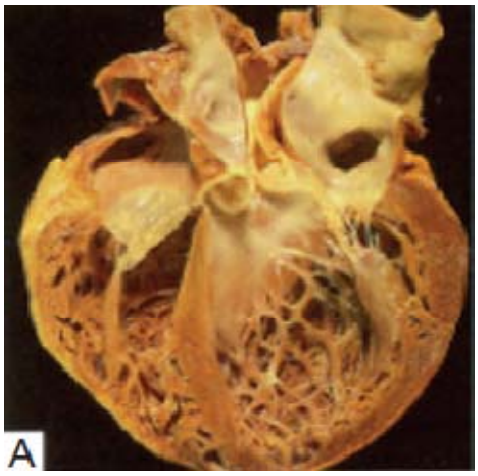
Pathology

Macroscopic Examination

Gross inspection of the heart demonstrates four-chamber enlargement. Most often, the ventricular walls are increased in thickness consistent with the myocyte hypertrophy that accompanies this disorder. Increasing chamber thickness is attributed to a compensatory mechanism aimed at reducing wall stress and is thus thought to play a beneficial role, averting further chamber remodeling.[19] The valvular structures themselves are normal, although chamber enlargement frequently leads to a dilation of the valvular orifice. Intracavitary thrombi are often noted and are preferentially located in the ventricular apices. The coronary circulation is most commonly normal, although the presence of non-occlusiveepicardial disease can raise a diagnostic conundrum wherein the

degree of cardiomyopathy is “out of proportion to the underlying coronary artery disease.”

A definition for ischemic cardiomyopathy has been arbitrarily set at a requirement for a greater than 70% stenosis in a major epicardial coronary artery, although pathologic studies have reported greater degrees of disease.[20] Preferential involvement of the right ventricle should suggest the diagnoses of arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) or corpulmonale (secondary to pulmonary hypertension).

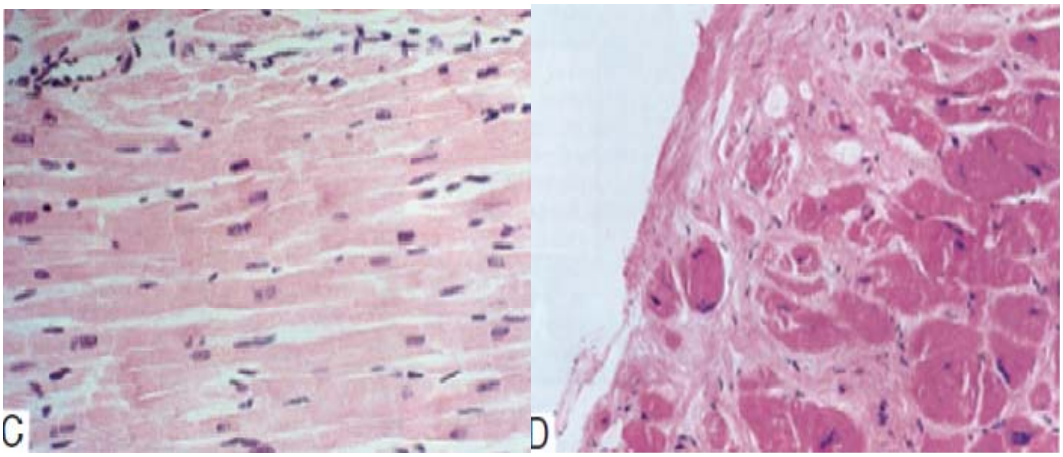


Gross Cardiac appearance showing four chamber enlargement

Histologic Examination

Histologic evaluation of the myocardium reveals varying degrees of myocyte hypertrophy and interstitial fibrosis.[20] Fibrosis most often affects the left ventricular sub endocardium or throughout the myocardium in interstitial or perivascular patterns. A finding of replacement fibrosis, an island of fibrotic tissue, often signifies a small area of tissue necrosis and suggests an ischemic etiology. It has been

difficult to identify characteristic immunologic or infectious findings; however, progress is being made, particularly with regard to viral persistence within the heart. Scattered cells considered to be lymphocytes are a frequent observation and may lead to a diagnosis of borderline myocarditis. This does not appear to affect prognosis.[21]



Cardiac myocytes hypertrophied Interstitial deposits of fibrotic tissue

Etiology

DCM accounts for approximately 25% of the cases of congestive heart failure in the United States.[22] The majority of the additional cases are due to specific cardiomyopathies, most notably ischemic or hypertensive cardiomyopathies, or nonsystolic heart failure.[23] The DCM phenotype can be manifested from specific systemic diseases or primary acquired processes, and intensive diagnostic evaluations in referral centres can reveal a specific associated cause of cardiomyopathy in ~50% of patients; the remaining 50% are assigned the diagnosis of exclusion, idiopathic DCM.[13] It is increasingly being appreciated that many of the

so-called cases of idiopathic DCM result from underlying genetic abnormalities or previous environmental insults that are difficult to detect at the time of clinical presentation. With the advent of sophisticated molecular and imaging technologies in clinical medicine, it is likely in the future that an increasing number of the idiopathic cases will have a specific diagnosis assigned.

Specific Cardiomyopathies with a Dilated Phenotype

Clinically, there are a host of important causes of secondary DCM that include alcohol and cocaine abuse,HIV infection,and metabolic abnormalities as well as the cardio toxicity of anticancer drugs,most notably doxorubicin and newly introduced drugs that inhibit tyrosine kinases (e.g., Herceptin and imitinab). The following four specific disorders are particularly important to recognize in that correct diagnosis has a major impact on patient management and chance for recovery.

Stress (Tako-tsubo or Broken Heart Syndrome)

An acute cardiomyopathy can be provoked by a stressful or emotional situation or exposure to high doses of catecholamines (sympathomimetic drugs).[24,25]This cardiomyopathy is most common among middle-aged women, appears to be related to catecholamine release, and in most cases is fully reversible with supportive care. Electrocardiographic findings of myocardial infarction in the presence of left ventricular dysfunction and absence of epicardial coronary stenosis

should prompt the diagnosis. Endomyocardial biopsy is of value to exclude myocarditis, which can also mimic acute myocardial infarction, and demonstrates contraction band necrosis.

Peripartum Cardiomyopathy

Peripartum cardiomyopathy[26,27]is defined as a cardiomyopathy manifesting between the last month of pregnancy and 6 months post partum. The etiology is unclear, but inflammatory factors are highly implicated, and some studies reveal a high incidence of lymphocytic inflammation. Peripartum cardiomyopathy is common in Africa but also is manifested in the developed world; it has an excellent long-term natural history if patients survive the initial period, during which time hemodynamic compromise may be severe[28]. Prognosis is worse in the developing world and among indigent patients in the United States[29].

It is important to differentiate peripartum cardiomyopathy from a chronic cardiomyopathy exacerbated by the volume load occurring during pregnancy[26]. Women who recover are at increased risk of recurrences with subsequent pregnancies; women with full recovery are more likely to tolerate a subsequent pregnancy than are those with. Peripartum cardiomyopathy leads to left ventricular systolic dysfunction are a form of DCM that results in signs and symptoms of heart failure. The criteria used for diagnosis of peripartum cardiomyopathy are as follows:

1. The development of heart failure in the last month of pregnancy or within 5 months of delivery
2. Absence of an identifiable etiology for the heart failure,
3. Absence of identifiable cardiac disease prior to the last month of pregnancy
4. Echocardiography demonstrates left ventricular systolic dysfunction.

Tachycardia-Induced Cardiomyopathy

Patients may develop a DCM with congestive heart failure in the face of recurrent or persistent tachycardias. The most common association is with atrial fibrillation or supraventricular tachycardia. There is a high rate of full recovery with control of the arrhythmia[30]. This cardiomyopathy is notable for the degree to which it resembles idiopathic DCM phenotypically, yet it is characterized by a remarkable degree of recovery in left ventricular function once the arrhythmia is controlled. Patients presenting with an atrial or supraventricular arrhythmia should undergo definitive therapy to control heart rate and to restore normal sinus rhythm.

Alcoholic Cardiomyopathy

For more than hundred years the toxic effects of alcohol have been known. Initially it was thought that the manifestations were due to nutritional deficiency rather than direct alcohol effects. Also additives in alcohol were found to be the cause of heart dysfunction in chronic alcoholics. The direct effects of alcohol leading to cardiomyopathy and also skeletal abnormalities were first described by Rubin et al. He

demonstrated that in a dose dependent manner alcohol is toxic to cardiac and striated muscle.[31,32]

An *alcohol cardiomyopathy* is diagnosed only when other causes of a dilated cardiomyopathy have been ruled out and there is a history of heavy and sustained alcohol intake. The dose requires to cause alcoholic cardiomyopathy is around 100g for several years. In susceptible individuals it is likely that even lower amounts of alcohol intake can produce cardiomyopathy. The histologic findings of alcohol cardiomyopathy are found to be nonspecific and that do not differ from idiopathic dilated cardiomyopathy. Other than history and examination the only probable distinguishing feature between idiopathic dilated cardiomyopathy and alcohol cardiomyopathy is that alcohol cardiomyopathy may present with a features of high cardiac output failure[31].

Diabetic cardiomyopathy

The Framingham Heart Study found that the frequency of cardiac failure is twice in diabetic men and five times more in diabetic women compared with age-matched control groups. Even after correction of hypertension, obesity, hypercholesterolemia, and coronary artery disease the diabetic cardiomyopathy still persists. The existence of diabetic cardiomyopathy in the presence of normal coronary arteries was first described by Rubler et al. Epidemiologic evidence of an association of a

specific DCM with diabetes became evident with the Framingham study[33,34].

The characteristic histopathological findings are fibrosis, which may beinterstitial, perivascular, or both. As the pathology progresses, there is increased myocyte loss and with replacement fibrosis. Common histopathologic findings of diabetic cardiomyopathy include small vessel disease, myocardial hypertrophy and interstitial fibrosis. The clinical features include symptoms and signs of heart failure in a patient with long standing diabetes mellitus. Diastolic dysfunction may be evidenteven much before systolic function is impaired. Management consists of treatment of congestive heart failure and glycaemic control.

Miscellaneous cardiomyopathies

HIV cardiomyopathy

The clinical, echocardiographic and also macroscopic findings of dilated cardiomyopathy in patients with HIV[35]was first described by Cohen and Colleagues. The mechanisms leading to dilated cardiomyopathy in HIV include

1. Post viral myocarditis: HIV itself can lead to cardiac dilatation and dysfunction. Viral infection causes an immune reaction to viral antigen that cross react with myocardial protein. Anti-myosin antibodies have been demonstrated in patients with HIV and cardiomyopathy. HIV infected monocytes may produce cytokines

which damages the cardiacmyocytes (innocent bystander mechanism)

2. Drug induced cardiomyopathy:Interferons,zidovudineetc can lead to cardiomyopathy.
3. Selenium andvitamin deficiency have also been implicated.
4. HIV patientswith concomitant cocaine abuse can also lead to dilated cardiomyopathy.
5. Other opportunistic infections liketoxoplasma,mycobacterium aviumintracellulare,cryptococcus,etc can also lead to cardiomyopathy. In advanced case of HIV cardiomyopathy is seen and in most patients biventricular failure is seen. For HIV cardiomyopathythere is no specific treatment.

Etiologic Basis for Idiopathic Dilated Cardiomyopathy

Rapidly advancing knowledge in four areas is shedding light on pathophysiologic mechanisms that may contribute to DCM and may in turn lead to new therapeutic approaches. These areas include (1) familial and genetic factors[36], (2) inflammatory and infectious factors, particularly viral infection[37], (3) cytotoxicity, and (4) cell loss and abnormalities in endogenous repair mechanisms[38,39].

Genetic and Familial Factors

Studies of the genetics of DCM offer major insights into the etiology of the disease. Two general lines of evidence initially suggested a genetic component to DCM. Familial studies indicated that in excess of 20% of patients with DCM had other family members with the condition, and conversely, certain inherited conditions, particularly muscular

dystrophies, had cardiomyopathy as a component. There are now abundant gene linkage studies with multiple genes identified; autosomal dominant and recessive as well as X-linked modes of inheritance exist[36].

A mutation in the gene encoding phospholamban implicates abnormalities in the excitation-contraction cascade as a cause of cardiomyopathy and supports attempts to treat cardiomyopathy with other elements of the calcium cycling machinery (i.e., delivery of the gene encoding the sarcoplasmic reticulum calcium pump [SERCA])[40].

The fact that genetic abnormalities play a role offers insights into the phenotype in general. Clearly, genetic predisposition may be a central factor in the development of primary and secondary DCMs. Genetic defects may be primary causes of DCM, or they may act as predisposing factors in the setting of an environmental stressor-host-environment interaction. Primary examples of the latter are viral infections and hypertension, wherein exposure may lead to DCM only in subpopulations of exposed individuals. Genetic predisposition may be of fundamental importance in the variable natural history of DCM and may contribute to responsiveness to therapy[41].

Knowledge of the genetics of DCM has led to the entry of genetic screening into the clinical arena and the development of specialty clinics at referral centres. Recent guidelines suggest that genetic screening and counselling should be considered in families in whom familial DCM is

suspected, as a means of early detection of cardiomyopathy in family members[36].

Inflammatory and Infectious Myocarditis

Myocarditis may result from viral (or other pathogen) infection, autoimmune disease, or a combination (autoimmune reaction stimulated by a viral infection)[42]. It is also increasingly possible that genetic factors increase the risk for development of cardiac disease after viral infection.

It has long been postulated that viral infection in susceptible hosts may be a proximate cause of cardiomyopathy and may serve as a precursor to the development of DCM. This hypothesis has been difficult to prove because of challenges in confirming viral infection in affected individuals coupled with the fact that common viruses are implicated in viral cardiomyopathy, leading to concerns of a high false-positive rate when viruses are detected in patients with heart failure. Lymphocytic myocarditis with or without myocyte necrosis has been considered the hallmark diagnostic finding necessary for a diagnosis, and criteria established for the histologic evaluation are termed the Dallas criteria[42].

Two general mechanisms for postviral cardiac injury have been invoked: autoimmune reactions and direct tissue injury resulting from viral infection of the heart. Both of these mechanisms are incompletely proved and remain controversial. The presence or absence of inflammation on endomyocardial biopsy, which varies greatly from study

to study, is used to substantiate immunologic injury. However, other studies have suggested different criteria (e.g., complement or immunoglobulin deposition). The postviral hypothesis has increasing support, and viral material has been detected on the basis of elevated viral titers, presence of viral genomic material by PCR, and detection of viral particles.

Autoimmunity

Studies support abnormalities of humoral and cellular immunity in DCM. Two general theories are proposed for an autoimmune cause of DCM: (1) viral components incorporate into the cardiac myocyte membrane, stimulating an antigenic response; and (2) anti–heart antibodies are generated as a result of myocardial damage as opposed to being the proximate cause. Certain specific human leukocyte antigen (HLA) class II antigens (particularly DR4) are associated with DCM. In addition, numerous circulating antimyocardial antibodies have been measured in DCM patients that react with a variety of antigens, including the myosin heavy chain, the beta adrenoceptor, the muscarinic receptor, sarcolemmal sodium-potassium adenosine triphosphatase, laminin, and mitochondrial proteins. A regimen of prednisone and azathioprine has recently been shown to improve ejection fraction in patients with virus-negative myocarditis[43].

Cytotoxicity and Deranged Intracellular Signalling

The direct action of various circulating factors is implicated in the pathophysiology of myocyte dysfunction. For example, tumor necrosis factor and endothelin levels are elevated in DCM. The exact role of these factors remains incompletely understood, and therapies to antagonize their effects have not been definitively established.

An additional molecular mechanism gaining increased experimental and clinical support is that of nitroso-redox imbalance, an intracellular phenomenon characterized by dysregulation of nitric oxide production coupled with increased production of reactive oxygen species[44].This imbalance is described in experimental animal models and in humans with DCM and causes cellular dysfunction and possibly cytotoxicity. Although not definitively proved, one mechanism postulated to explain the response of DCM patients to hydralazine–isosorbidedinitrate is a restoration of nitroso-redox balance.

Injury, Cell Loss, and Endogenous Repair

A variety of other causes related to damage to cellular constituents of the heart are proposed as etiologic factors. Although none is accepted as the absolute cause, the variety of mechanisms highlights the notion of a final common pathway, with various insults converging on a set of mechanisms that all result in a common phenotypic response to injury. Many of the mechanisms, such as endocrine disturbances and toxic

exposures, derive from the existence of specific examples of secondary cardiomyopathies.

Ischemia due to hyperreactivity or spasm of the microvasculature may contribute to diffuse myocyte necrosis and replacement fibrosis. The classic disorder in which this is manifested is scleroderma heart disease. Increased myocyte apoptosis is described in DCM and ARVD/C, leading to the suggestion that augmented cell loss may contribute to the development of left ventricular remodeling in DCM processes. Although there are an increasing number of experimental studies supporting cardiac recovery when antiapoptotic agents are administered in animal models[45], the exact role of apoptosis in these conditions is not known. Further, the role of cell loss in DCM has become more interesting in light of recent accumulating data supporting the idea that endogenous cardiac stem cells repopulate cardiac myocytes throughout life[39], thereby serving a homeostatic balancing mechanism for ongoing cell loss and cell replacement after tissue injury. Indeed, studies already support the idea of cardiac stem cell senescence contributing to the development of human cardiomyopathy[38]. Thus, depletion or dysfunction of endogenous cells with capacity to divide and to differentiate in cardiac cellular constituents may be a central pathophysiologic contributor to cardiomyopathic processes.

Clinical Evaluation of the Dilated Cardiomyopathies

History

DCM affects individuals of all ages, including neonates and children[46,47]. In adults, the incidence of DCM is estimated to be between 5 and 8 per 100,000 persons per year. DCM is most frequent in middle age and affects men to a greater degree than women. Although the incidence of ischemic cardiomyopathy is higher than that of DCM, these two diagnoses account for an equal number of heart transplantations performed.

In the case of DCM, the clinical presentation of patients can vary substantially. In some patients, symptoms develop very gradually and diagnosis can result from the detection of cardiomegaly on routine chest radiography. Intercurrent illnesses frequently precipitate congestive heart failure in individuals with DCM. A significant minority of patients with DCM present with aggressive, life-threatening congestive heart failure (fulminant heart failure) that can require the most intensive forms of mechanical intervention[21]. The causes of the fulminant presentation vary from idiopathic cardiomyopathy to fulminant lymphocytic myocarditis to giant cell myocarditis[18,22]. The determinants of these various forms of clinical presentation are poorly understood.

Evaluation for Secondary Cardiomyopathies

An initial history must focus on identifying etiologic factors[13]. A past or associated history of rheumatologic, endocrine, or infectious diseases or of previous neoplasia should be sought.In patients with a history of cancer, treatment with anthracyclines, tyrosine kinase inhibitors, or irradiation is particularly relevant. The family history can often reveal heritable forms of cardiomyopathy. Patients should be questioned about the consumption of alcohol, tobacco, and illicit drugs. Travel history can reveal exposure to geographically related infectious pathogens.

The most typical symptoms are those of congestive heart failure and include dyspnoea, fatigue, and volume gain. A minority of patients report chest pain, which can signify epicardialcoronary disease, subendocardial disease, or pulmonary embolism. A report of abdominal discomfort or anorexia is frequent in late stages of the disease and suggests hepatomegaly or bowel edema, respectively.

Common late complications include thromboembolic events, which may be systemic, originating from dislodgment of left atrial and ventricular intracardiac or pulmonary thrombi from the lower extremity venous system.

Physical Examination

Particular attention should be paid in the physical examination to excluding findings of valvular heart disease. S_3 and S_4 gallops are invariably present in DCM. The S_3 must be differentiated from a pericardial knock or an opening snap of mitral stenosis, both of which are higher pitched sounds than the S_3. Patients with fulminant heart failure of new onset will frequently be tachycardic and will develop a gallop rhythm in which S_3 and S_4 fuse. Attention should be paid to differentiation of right-sided gallops and murmurs to consider the possibility of right-sided involvement.

NEWYORK HEART ASSOCIATION CLASSIFICATION

Functional Capacity	**Objective Assessment**
Class I	Patients with cardiac disease but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitations, dyspnoea, or anginal pain.
Class II	Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnoea, or anginal pain.
Class III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnoea, or anginal pain.
Class IV	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

American College of Cardiology/American Heart Association Stages of Heart Failure

Stage	Definition	Patient Description
A	High risk for developing heart failure (HF)	Hypertension Coronary artery disease Diabetes mellitus Family history of cardiomyopathy
B	Asymptomatic HF	Previous myocardial infarction (MI) LV hypertrophy or systolic dysfunction Asymptomatic valvular disease Known structural heart disease
C	Symptomatic HF	Shortness of breath and fatigue Reduced exercise tolerance
D	Refractory end-stage HF	Marked symptoms at rest despite maximal medical therapy (e.g., those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions

Non-invasive Evaluation

For patients presenting with DCM, the initial evaluation should focus on identification of reversible and secondary causes. Even though the presentation of the patient with a dilated ventricle and heart failure may be fairly uniform, a wide array of specific and secondary cardiomyopathies may cause a clinical presentation of a DCM. The first step in the diagnostic evaluation involves screening biochemical testing, including serum electrolytes, phosphorus, calcium, and markers of renal function (serum creatinine and urea) [13]. Endocrine function should be screened, notably thyroid function (hyperthyroidism and hypothyroidism) and possibly urinary evaluation of catecholamine levels to exclude

pheochromocytoma. To screen for rheumatologic conditions, an antinuclear antibody and erythrocyte sedimentation rate should be obtained. When suspected, rarer causes of cardiomyopathy can be excluded with blood testing. For example, Lyme titres can be a useful screen for Lyme carditis. Iron studies may assist in evaluating hemochromatosis, and HIV testing is valuable.

The use of biomarkers (such as troponin) to assess myocardial necrosis and the use of circulating brain natriuretic peptide (BNP or pro-BNP) levels may serve as useful adjunctive strategies to help determine diagnosis or prognosis. Further, there is increasing support for the use of serum uric acid levels as a prognostic marker[14,48]. A chest radiograph offers supporting evidence for the diagnosis and in some cases is the initial mode of detection. Cardiomegaly may be appreciated, as may evidence of pulmonary vascular redistribution. Rarely, interstitial and alveolar oedema is present on initial presentation. With advancing heart failure, pleural effusions are present, and dilated azygos veins and superior vena cava indicate right-sided volume overload.

Electrocardiography

There are no specific electrocardiographic findings signifying DCM. Sinus tachycardia is often present in proportion to the degree of heart failure. Typical changes in the QRS complex include poor R wave progression, intraventricular conduction delays, and left bundle branch

block. A wide QRS complex portends a worse prognosis and has now emerged as a clinical indicator of responsiveness to cardiac resynchronization therapy. Patients with substantial left ventricular fibrosis may exhibit anterior Q waves even in the absence of a discrete scar or epicardial coronary artery obstructions.

A broad array of abnormalities may be manifested, such as nonspecific ST-segment and T wave abnormalities as well as P wave alterations, notably left atrial abnormality. Nonsustained ventricular tachycardia is extremely common on 24-hour ambulatory monitoring and represents a predictor of all-cause mortality. Persistent supraventricular or ventricular tachyarrhythmia's represent an important etiologic factor for ventricular dysfunction, [30] and restoration of sinus rhythm or heart rate control may lead to recovery of ventricular function. Control of atrial fibrillation is also important because of atrial transport issues contributing to cardiac output. In addition, atrial fibrillation should prompt consideration of tachycardia-induced cardiomyopathy.

Echocardiography

Echocardiography is a cornerstone in the evaluation and management of patients with DCM. Two-dimensional echocardiography is a highly useful and readily available technique to assess ventricular size and performance and to exclude associated valvular or pericardial abnormalities. Doppler echocardiography permits the evaluation of

valvular regurgitation or stenosis and the quantification of cardiac output. Doppler detection of restrictive filling patterns may indicate disease of greater severity.

Pericardial effusion may be present. Performing echocardiography during dobutamine stimulation may identify occult coronary artery disease by provoking regional wall motion abnormalities, differentiating these patients from those with idiopathic DCM. Moreover, significant contractile reserve during dobutamine infusion represents a positive prognostic finding. Three-dimensional echocardiography may be of additional value in assessing mitral valve orifice remodelling and determining ventricular dyssynchrony.

Radionuclide Imaging

Nuclear imaging protocols for myocardial perfusion stress imaging may be useful to exclude an ischemic cause of dilated heart failure. Radionuclide ventriculography also provides evidence of cardiac structure and function, showing increased chamber volumes at end diastole and end systole; it provides quantification of reduced ejection fraction in either or both ventricles, and it can elucidate the regional nature of wall motion abnormalities. Not always necessary, this technique can be of particular value if echocardiography is technically suboptimal.

Cardiac Magnetic Resonance Imaging and Multidetector Computed Tomography

Cardiac magnetic resonance imaging (CMR) and multidetector computed tomography are relatively new imaging modalities that are likely to become increasingly useful to evaluate patients with cardiomyopathies[49]. Specific cardiomyopathic disorders in which CMR has proved particularly valuable include ARVD/C[50], endocardial fibroelastosis, myocarditis[51], amyloidosis[52], and sarcoidosis. CMR evaluation is also emerging as a critical tool to understand DCM pathophysiology and may contribute to identification of patients at particular risk for complications, such as sudden cardiac death (e.g., within DCM subsets, those with or without areas of replacement fibrosis that may predispose to electrical instability and sudden cardiac death) [53]. CMR is also emerging as an important tool in the delineation of infiltrative and inflammatory cardiomyopathies.

Invasive Evaluation Including Endomyocardial Biopsy

Catheterization for the exclusion of epicardial coronary disease is essential in the management of the patient presenting with DCM. Because DCM and heart failure increase the false-positive and false-negative rates of non-invasive nuclear assessment for myocardial ischemia, performance of coronary angiography is often necessary to exclude epicardial coronary obstructive disease[20]. It is increasingly relevant to obtain hemodynamic

assessments in individuals presenting with acute or worsening heart failure. Use of these diagnostic tests is currently nonuniform[54]. Catheterization usually reveals elevated left ventricular end-diastolic and pulmonary artery wedge pressures. Pulmonary arterial hypertension may be of variable degrees, ranging from mild to severe. The right ventricle is frequently involved and enlarged, hemodynamically manifesting with increased right ventricular end-diastolic, right atrial and central venous pressures.

Left ventriculography demonstrates varying degrees of ventricular dilation and diffuse chamber hypo kinesis. There may be a degree of regionality to the decreased function resembling ischemic heart disease, although a diffuse pattern is frequently present. It is not always possible to distinguish between left ventricular dilation due to severe mitral regurgitation associated with primary mitral valve disease and DCM with secondary mitral regurgitation.

Coronary arteriography is particularly important to exclude coronary obstructive disease. In patients with DCM, the arterial circulation is typically normal although vasodilator function may be abnormal.

Biopsy

The role of endomyocardial biopsy to evaluate the myocardium histologically has been historically controversial in the evaluation of the

patient presenting with structural heart disease or symptoms of heart failure[55,56]. Recently, however, expert guidelines have been published that offer significant guidance as to the indications for endomyocardial biopsy[18]. This procedure, which is routine in the management of heart transplant recipients, allows the acquisition of small pieces of myocardium by use of a flexible bioptome. Currently available bioptomes are advanced transvenously, most commonly by a right internal jugular venous approach, to the right ventricular septum. If it is required, the left ventricular septum may be sampled by a trans arterial approach. This procedure is currently performed with either fluoroscopic or echocardiographic guidance. Although not reported in the literature, the widespread use of disposable bioptomes, which have replaced reusable Stanford-Caves devices, has led to a reduction of complications, particularly right ventricle perforation.

Perhaps the most compelling reason in favour of routine biopsy is the detection of a few relatively rare diseases in which accurate diagnosis yields a life-threatening disease with specific management[21,51]. For example, lymphocytic and giant cell myocarditis must be detected early in the course of the presentation for patients to survive and can be separated from each other only by histologic evaluation[43,58]. Biopsy is also an established method for grading the severity of anthracycline cardiomyopathy and has potential similar value for cardiac amyloidosis.

A biopsy finding that is negative for inflammation is also valuable in patients with rapidly progressive severe decompensated heart failure, insofar as it may prompt advancement to aggressive mechanical support earlier in the patient's clinical course. Whereas widespread use of the myocardial biopsy is no longer routinely recommended, recent guidelines and treatment trials continue to add clarity around appropriate selection of patients. In patients with fulminant heart failure, particularly those with new-onset cardiomyopathy, the risk-benefit assessment is more clearly in favour of performing a biopsy to more rationally allocate patients for emergent heart transplantation listing or for insertion of a mechanical assist device.

Patients who have fulminant lymphocytic myocarditis have excellent long-term prognosis after short-term hemodynamic support[21]; those with giant cell myocarditis should be aggressively immunosuppressed or listed for heart transplantation[58]; and those with idiopathic cardiomyopathy (suggested by the absence of myocardial inflammation on biopsy) should be aggressively supported and converted to conventional therapy once stabilized.

Management

Pharmacologic and Device Therapy

Whereas the concept of specific etiology-based therapies represents an ongoing quest for patients with DCM, the general treatment of these

patients should follow the practice guidelines for all patients with heart failure[59]. Similarly, the use of prophylactic implantable cardiac defibrillators and biventricular pacemakers is indicated in appropriate patients with nonischemic and ischemic DCM [59,60].

Surgery

Patients with valvular heart disease, coronary artery disease, pericardial disease, or congenital heart defects should have these conditions corrected surgically, when appropriate. Other specific operations geared toward the cardiomyopathic heart include approaches motivated by the concept of restoring chamber geometry or interventions to provide mechanical support. Approaches to achievement of reverse remodelling surgically include left ventricular reconstruction and implantation of external restraint devices. Left ventricular assist devices provide aggressive mechanical support to patients with advanced decompensated heart failure.

Emerging Specific Therapies

Only recently are specific etiology-based therapies being evaluated. These include agents to eradicate persistent viral infections and immunomodulatory agents. Stem cells for cardiac regeneration and gene therapy approaches are in clinical trials.

MATERIALS AND METHODS

Place Of Study : Institute of internal medicine, RGGGH, MMC.

Study Design : Cross sectional study

Ethical Committee Clearance : Obtained

Period of Study: May 2011 to Oct 2011

SELECTION CRITERIA

Inclusion Criteria

1. Clinical criteria:

   Patients with symptoms and signs of heart failure.

2. ECHO criteria:

   Left ventricular ejection fraction < 45%

   Global hyokinesiaof LV

   Dilatation of all the chambers of heart

   Left ventricular end diastolic dimension > 3 cm / body surface area.

Exclusion criteria

1. Pericardial disease
2. Corpulmonale with CHF.
3. Hypertrophic cardiomyopathy
4. Restrictive cardiomyopathy
5. Congenital heart disease

STUDY POPULATION

The subjects for the study were selected from cases admitted to the medical wards of Government General Hospital, Chennai during the period from May 2011 to Oct 2011 who fitted in the criteria described above. The diagnosis of dilated cardiomyopathy was made on the basis of history, physical findings and echocardiographic features.

STUDY DESIGN: Cross sectional study

METHODOLOGY

100 representative cases of dilated cardiomyopathy which fitted in the criteria were selected. A detailed history was obtained from them and symptom analysis was done. A detailed clinical examination was also done.

A 12 lead electrocardiogram was obtained and analysed.

A chest radiograph which comprised of a posteroanterior chest film was obtained. In all cases the cardio thoracic ratio, pulmonary infiltrates, pulmonary plethora, pleural effusion was looked for.

Echocardiogram was done for all patients. In all patients chamber dimension, EF, global hypokinesia were looked for and the results are interpreted.

The diagnosis of ischemic DCM was based on either past history of myocardial infarction or coronary angiography demonstrates significant luminal occlusion (>70%).

Peripartum cardiomyopathy was diagnosed by using criteria laid down by Demakis et al which includes (1) Development of heart failure in the last month of pregnancy or within 5 months of delivery (2) absence of identifiable heart disease prior to the last month of pregnancy. (3) Echocardiogram demonstrates classical left ventricular dysfunction (4) Absence of other identifiable causes of heart failure.

The diagnosis of diabetic cardiomyopathy was made in patients with long standing (>10 years) diabetes mellitus and in whom no other cause was obvious.

Similarly patients with echocardiography proven dilated cardiomyopathy with history oflong term (> 10 years) alcohol consumption in whom no other causes were found were diagnosed as alcoholic cardiomyopathy.

If no obvious cause was found they are categorized as idiopathic DCM.

The clinical profile along with the probable etiology, radiological and electrocardiographic findings were summarised and compared with existing data.

COMPETING INTEREST: NIL

FINANCIAL SUPPORT: NIL

OBSERVATIONS AND RESULTS

Out of these 100 patients 57 were male patients and 43 were females. The age of patients ranged from 18 to 76 years.

AGE

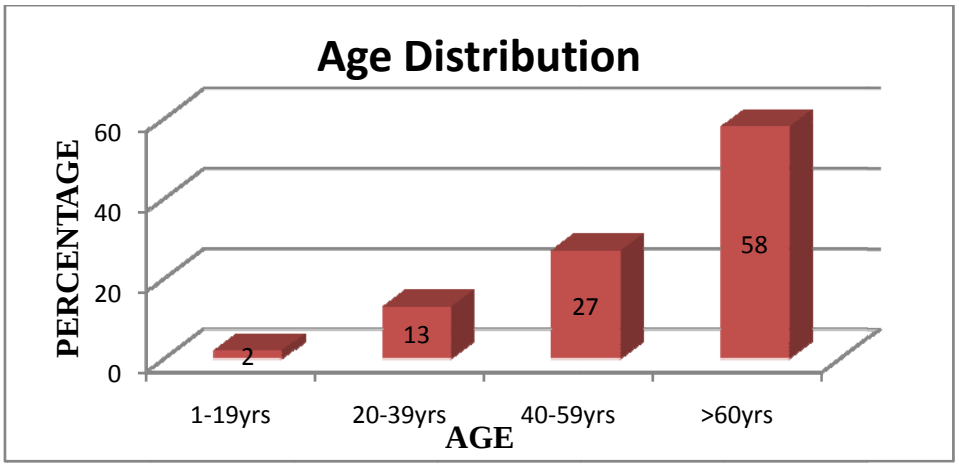
In our present study the following age distribution was observed. The distribution shows that the peak incidence was above 6th decade.

Age distribution is shown in the table below.

TABLE 1: AGE DISTRIBUTION

Age groups	Number of case	Percentage
1-19	2	2
20-39	13	13
40-59	27	27
>60	58	58

FIGURE 1: GRAPH SHOWING AGE DISTRIBUTION



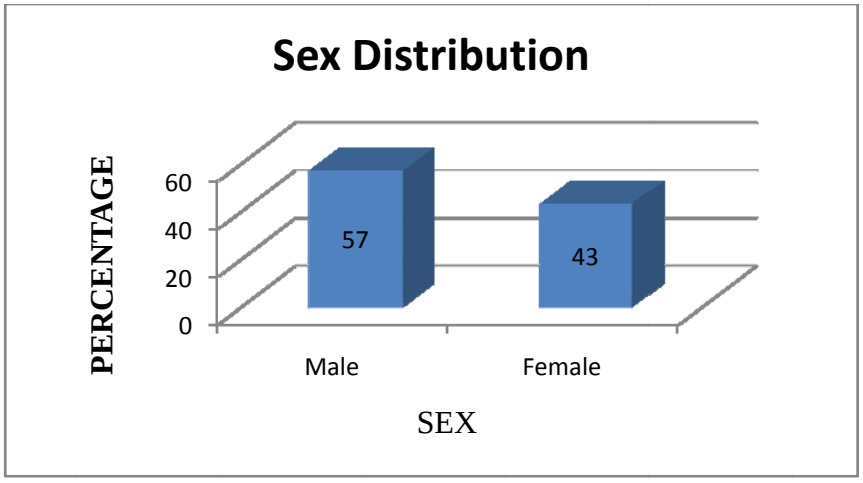
SEX

In our study 57 patients were males and 43 patients were females.

TABLE 2: SEX DISTRIBUTION

Sex	Cases	Percentage
Male	57	57
Female	43	43

FIGURE 2: GRAPH SHOWING SEX DISTRIBUTION



ALCOHOL HABITS

In our study 36 males were alcoholic. Among these 15 of them used to take alcohol for more than 10 years, they also consumed more than 100g/day. The remaining 21 of them used to take alcohol less than twice a week, the duration is also less than 10 years and the quantity consumed is also less than 40g/day.

TABLE 3: ALCOHOL DURATION

Alcohol duration	No. of cases	Percentage
< 10 Yrs	21	21
10-20 Yrs	9	9
>20 Yrs	6	6

PAST HISTORY OF MYOCARDIAL INFARCTION

Past history of myocardial infarction present in 48 among 100 patients. Among them documented ECG & ECHO records were available with thirty four of them. Only twenty one of them are under regular treatment. Eighteen of them are alcoholic & twenty four of them are smoker.

SYMPTOMATOLOGY

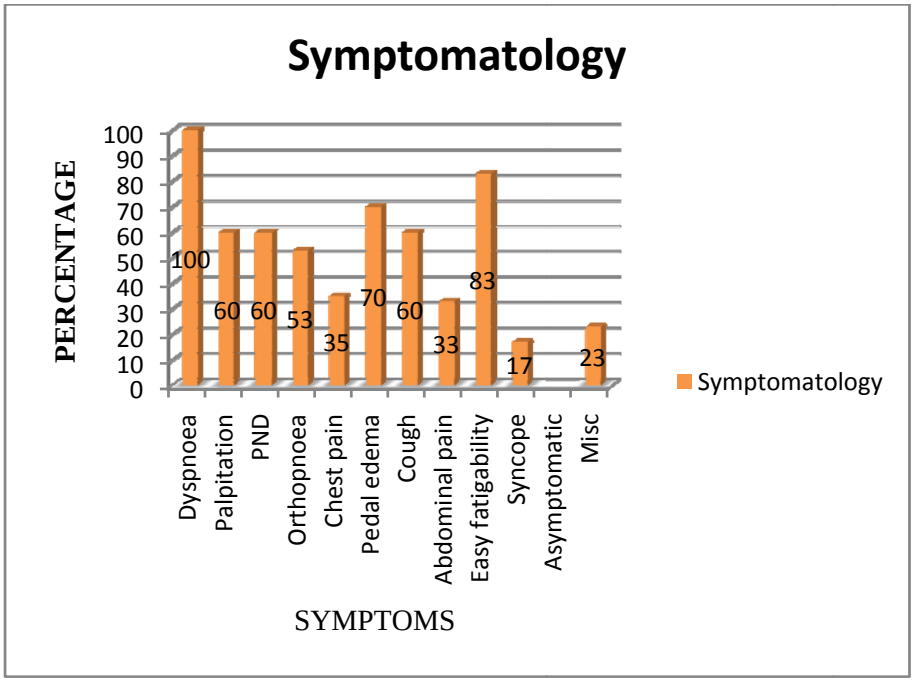
All the patients in this study presented with exertional dyspnea. Easy fatigability was observed in 83 % of subjects constituting the next most common symptom followed by pedal edema in 70 % of patients. History of paroxysmal nocturnal dyspnea,cough and palpitation were seen in 60 % of subjects followed by orthopnea 53 %, chest pain 35 %, abdominal pain 33 % and syncope 17 %.

The details about the symptoms are depicted in the table

TABLE 4: SYMPTOMATOLOGY

Symptoms	No. of cases	Percentage
Dyspnea	100	100
Palpitation	60	60
PND	60	60
Orthopnea	53	53
Chest pain	35	35
Pedal edema	70	70
Cough	60	60
Abdominal pain	33	33
Easy fatigability	83	83
Syncope	17	17
Asymptomatic	0	0
Misc	23	23

FIGURE 3: GRAPH SHOWING SYMPTOMATOLOGY



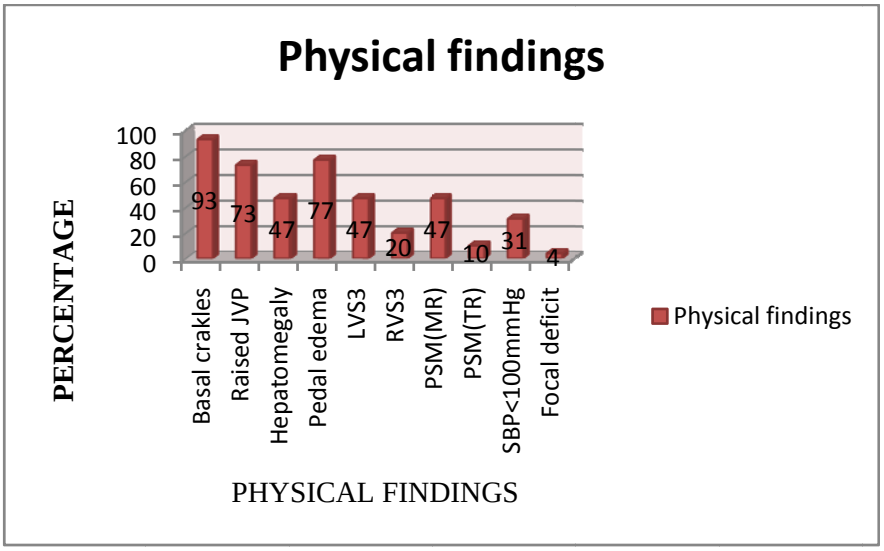
PHYSICAL FINDINGS

Basal crackles were seen in about 93 % of the subjects. Pedal edema wasseen in 77 %. Raised JVP was present in 73.3% and hepatomegaly in 47 %. Apicalpan systolic murmur was noticed in 47 % with LVS3 seen in 47 %. Pan systolic murmurin tricuspid area (TR) was present in 10% while RVS3 was present in 20% of our patients.Systolic blood pressure < 100 mmHg was observed in 31 % and four patients had stroke.

TABLE 5: PHYSICAL FINDINGS

Signs	No. of cases	Percentage
Basal crackles	93	93
Raised JVP	73	73
Hepatomegaly	47	47
Pedal edema	77	77
LVS3	47	47
RVS3	20	20
PSM(MR)	47	47
PSM(TR)	10	10
SBP<100 mmHg	31	31
Focal neurological deficit	4	4

FIGURE 4: GRAPH SHOWING PHYSICAL FINDINGS



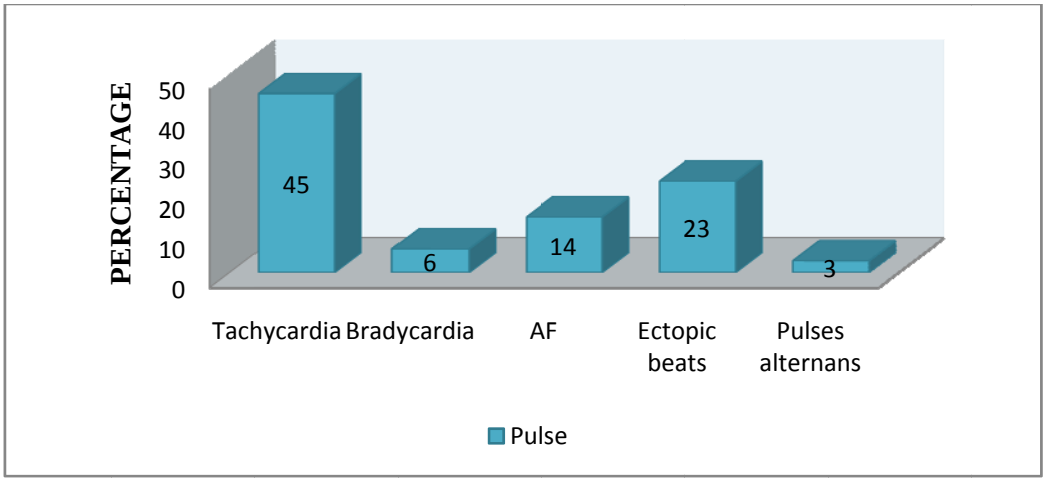
PERIPHERAL PULSE

Abnormalities of peripheral pulse included, bradycardia,tachycardia ectopic beats, pulsusalternans andatrial fibrillation. Ectopic beats were present in 23 %, tachycardia observed in 57 % and atrial fibrillation seen in 14 % of patients. Bradycardia was seen in 6% and pulsusalternans was seen in 3 % of subjects.

TABLE 6: PERIPHERAL PULSE

Pulse	No. of cases	Percentage
Tachycardia	45	45
Bradycardia	6	6
AF	14	14
Ectopic beats	23	23
Pulses alternans	3	3

FIGURE 5: GRAPH SHOWING PERIPHERAL PULSE



RADIOLOGICAL FEATURES

Almost all patients showed cardiomegaly in chest X-ray. The cardio thoracic ratio was more than 0.7 in 12 %, it was between 0.6 to 0.7 (moderate) in 46 %and 42 % of patients had mild cardiomegaly i.e. between 0.5 to 0.6. Pulmonary plethora was observed in 51 % of subjects while pleural effusion was noticed in 27 % of patients.

TABLE 7: RADIOLOGICAL FEATURES

Chest X ray		No. of cases	Percentage
CT Ratio	50-60%	42	42
	60-70%	46	46
	>70%	12	12
Pleural effusion		27	27
Pulmonary plethora		51	51

ELECTROCARDIOGRAM

The electocardiographic profile includes abnormalities of rate, rhythm, axis and chamber enlargement. The most common abnormality noticed was ventricular ectopics seen in 46 % of patients. Sinus tachycardia was seen in 34 % of subjects. Left bundle branch block was seen in 42 % of patients. Right bundle branch block was seen in 13 %. Non specific ST-T changes were noticed in 29 % whereas AF was present in 14 %. Left ventricular hypertrophy was seen in 22 % and LAE in 16 % of subjects. Complete heart block was present in only 3 patient (3 %). The axis was almost normal in majority of patients. Left axis deviation was observed in 16 % and right axis deviation in 6 %.

TABLE 8: ECG CHANGES

Parameters		n	%
QRS Axis	Normal	74	74
	Left axis deviation	17	17
	Right axis deviation	9	9
Arrhythmias	Sinus tachycardia	45	45
	Atrial ectopics	11	11
	AF	14	14
	SVT	7	7
	Ventricular ectopics	46	46
	VT	4	4
	CHB	3	3
	LBBB	42	42
	RBBB	13	13
ST-T Changes		29	29
Atrial enlargement	LAE	16	16
	RAE	6	6
Ventricular hypertrophy	LVH	22	22
	RVH	9	9
	BOTH	6	6

ECHOCARDIOGRAPHY

The mean left ventricular ejection fraction was found to be 30.87 %. The LV ejection fraction was less than 20% in 6% of patients. It was between 20-29% in 40 %, between 30-39% in 37 % of patients and between 40 to 45% in 17 % of patients. The mean LVEDD was 6.04 ± 0.74 cm with majority i.e. 54 % ofsubjects having LV end diastolic diameter more than 6 cm. The mean LVESD was 4.92 ± 0.62cm; with majority of patients (47 %) having end systolic diameter more than 5 cm. Global hypokinesia and dilatation of all 4 chambers were seen in

almostall the patients. In our study 68 % of patients had mitral regurgitation, 8 % of patients had tricuspid regurgitation and 9 % of patients had pericardial effusion.

TABLE 9: ECHO CHANGES

Parameter	Range	No. of cases	%
EF	40-45%	17	17
	30-39%	37	37
	20-29%	40	40
	<20%	6	6
LVEDD	4.5-4.9cm	13	13
	5.0-5.9cm	33	33
	>6cm	54	54
LVSD	3.5-4cm	20	20
	4-4.9cm	33	33
	>5cm	47	47
MR		68	68
TR		8	8
AR		4	4
Pericardial effusion		9	9

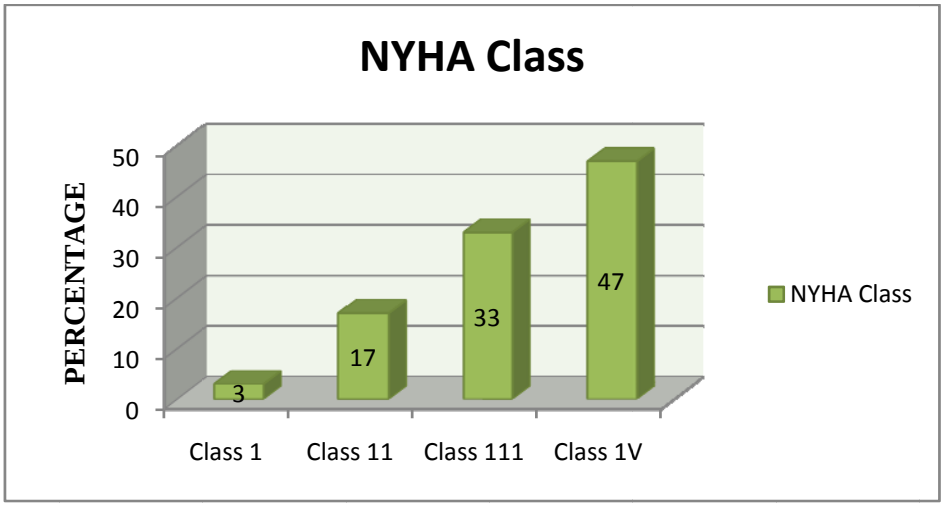
NYHA CLASS

Majority of the patients in our study were in NYHA class III (33%) and class IV (47%) group.

TABLE 10: NYHA CLASS

NYHA Class	No. of cases	Percentage
Class 1	3	3
Class 11	17	17
Class 111	33	33
Class 1V	47	47

FIGURE 6: GRAPH SHOWING NYHA CLASS



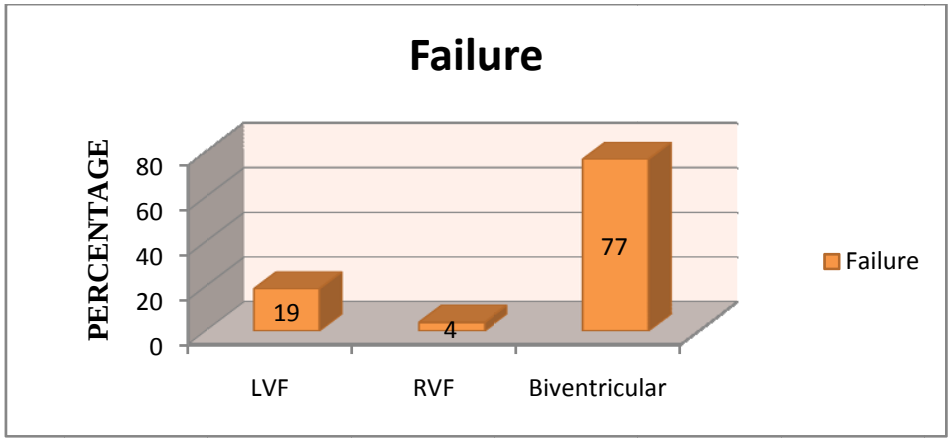
HEART FAILURE

Biventricular failure was present in 77 % of patients and isolated LV failure was seen in19 %. 4 % of patients in our study had RV failure.

TABLE 11: HEART FAILURE

Heart failure	No. of cases	Percentage
LVF	19	19
RVF	4	4
Biventricular	77	77

FIGURE 7 : GRAPH SHOWING HEART FAILURE



CAUSES OF DILATED CARDIOMYOPATHY

The most common type of dilated cardiomyopathy was found to be ischemic dilated cardiomyopathy in our study comprising 47 % of all cardiomyopathies followed by alcoholic cardiomyopathy (15 %) and diabetic cardiomyopathy (11 %).Idiopathic DCM was seen in 12 % of subjects while peripartum cardiomyopathy was seen in 9 %. Miscellaneous group included 6 cases (6 %),which includes three cases ofvalvular cardiomyopathy (mitral regurgitation & aortic

regurgitation),two cases of HIV cardiomyopathy and one post viral myocarditis.

TABLE 12: CAUSES OF DILATED CARDIOMYOPATHY

Cause	No.of Cases	Percentage
Ischemic	47	47
Alcoholic	15	15
Idiopathic	12	12
Diabetic	11	11
Peripartum	9	9
Miscellaneous	6	6

FIGURE 8: GRAPH SHOWING ETIOLOGY

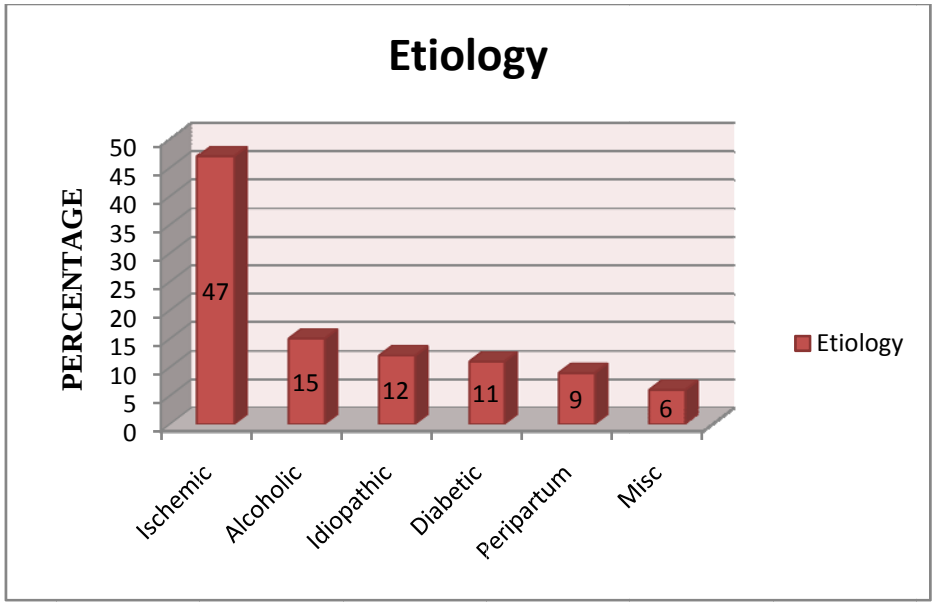


FIGURE 9: GRAPH SHOWING ETIOLOGY

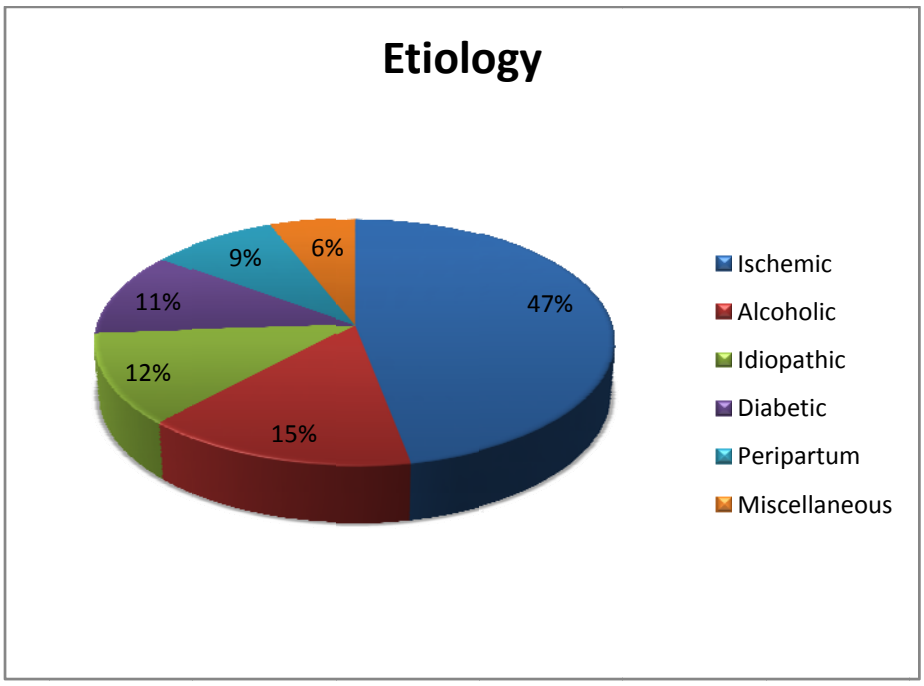


TABLE 13: ALCOHOL INTAKE AND DCM

Alcohol intake	Alcoholic DCM	Non alcoholic DCM	Total
Present	15	21	36
Absent	0	64	64
Total	15	85	100

FIGURE 10: GRAPH SHOWING ALCOHOL AND DCM

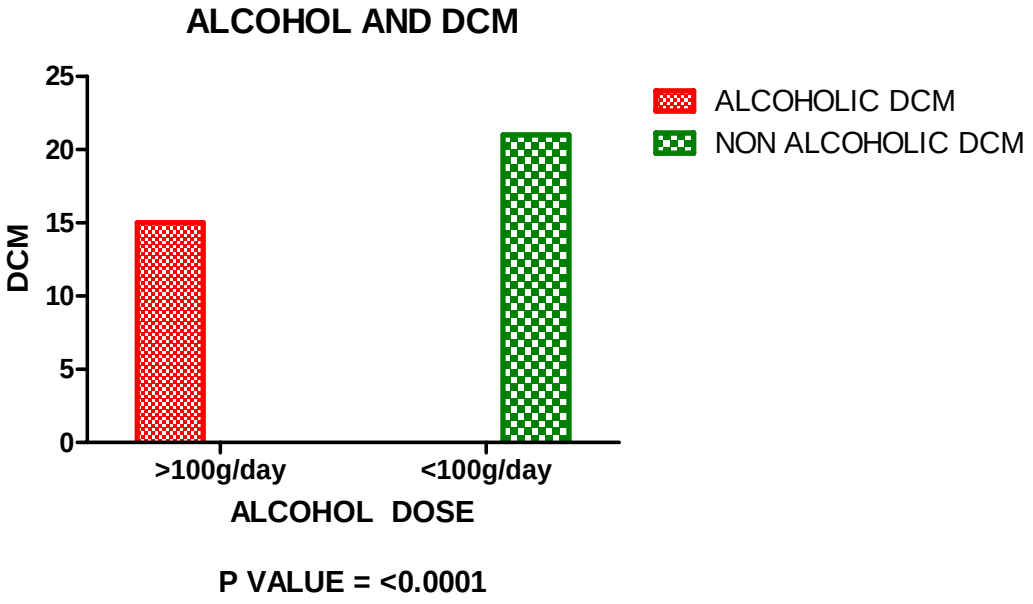


TABLE 14: COMPARISON OF EF & LVEDD AMONG ISCHEMIC AND NONISCHEMIC DCM

Parameter	All Cases	Ischemic DCM	Non Ischemic DCM	P value
Mean EF	31.26 ±7.59	31.10±8.44	31.40±6.83	0.85
Mean LVEDD	6.044±0.75	6.07±0.79	6.02±0.71	0.72

P value calculated using Fisher exact test.

DISCUSSION

Among 100 patients in our study 57% were found to be males and the remainder were females. In males, dilated cardiomyopathy was most commonly seen in the elderly males (mean age 56.6±12.5 years).In females DCM was predominantly seen in middle age. The underlying etiology varied with the age group. In one study the mean age was 52.9 ± 15.1 years in males and 51.3.9±17.7 years in females. In another study the mean age was 64.4 years in males and 55.5 years in females. In a study done in 2004, the mean age of presentation was found to be 42.6±9.1 years, with males comprising 73.6% and females comprising 26.4% of the study population.

Symptomatology

The most common presentation in our study was found to be biventricular failure which was seen in 77 % of cases. Isolated left ventricular failure was seen in 19 % of patients, most of them were ischemic DCM. Predominant right ventricular failure was seen in two patients with alcohol cardiomyopathy. Most of the patients were in NYHA class IV (47 %) and class III (33 %) while 17 % were in NYHA class II. Dyspnoea was the commonest symptom found in almost all the patients. Paroxysmal nocturnal dyspnoea was seen in 60 patients (60%) while orthopnoea was noticed in 53 patients (53 %).

Cough

Cough was present in 60% of our patients probably due to pulmonary congestion. Six patients (i.e. 6 %) in our study had respiratory infection like acute bronchitis and bronchopneumonia.

Easy fatigability

Easy fatigability was the second most commonest symptom found in 83 % of our subjects. It was noticed more commonly in patients with biventricular failure and less commonly in patients with isolated left ventricular failure. Easy fatigability was attributed mainly to chronic heart failure itself in most cases. In addition factors like anaemia, cardiac cachexia, etc. contribute to easy fatigability.

Pedal edema

Pedal edema was seen in majority (70%) of our patients. Pedal edema was the predominant symptom in patients with Alcoholic cardiomyopathy and idiopathic DCM. Anasarca was noticed in 11 patients (i.e. 11 %) of which 5 had alcoholic DCM, 5 had ischemic DCM, while 1 had idiopathic DCM.

Abdominal pain

33% of patients in our study had abdominal pain. Abdominal pain was attributed to hepatic congestion. The other possible mechanism for abdominal pain in these patients could be gastritis.

Palpitation

Palpitation was noticed in 60 patients (i.e. 60 %) in our study. Sinus tachycardia was seen in 57 % of patients secondary to chronic heart failure. Palpitation was also attributed due to atrial fibrillation, atrial / ventricular ectopics, and supra ventricular tachycardia etc.

Chest pain

In 35 % of our patients chest pain was present. Most of these patients had ischemic DCM. The cause of chest pain in these patients was found to be due to chronic myocardial ischemia.

Syncope

Syncope was present in 17 % of our patients. Syncope is mainly secondary to low cardiac output in most of the cases.

Symptomatology	**Our study (%)**	**Ahmad et al (%)[60]**	**Jain et al (%)[61]**
Dyspnoea	100	96.3	100
Easy fatigability	83	83.6	80
Pedal edema	70	56	100
PND	60	36.3	53
Cough	60	56.3	52
Palpitation	60	65.4	66.7
Orthopnoea	53	40	100
Abdominal pain	33	41.8	30
Syncope	17	1.8	2

Physical signs

Peripheral pulse

Tachycardia was present in 45 % of patients. Bradycardia was present in 6 patients secondary to complete heart block. Atrial fibrillation was noticed in 14 % of patients. Ectopic beats was present in 23 % of patients in our study. Pulsus alternans was seen in three patients.

Signs of left heart failure

Basal crackles were present in 93 % of our patients in our study. Basal crackles were attributed mainly to pulmonary edema / pulmonary congestion secondary to LV failure.

Cardiac examination revealed LVS3 in 47 % of our patients in this study. Pan-systolic murmur secondary due to mitral regurgitation were seen in 47 % of our patients. RVS3 was found in 20% of patients while PSM in left parasternal area secondary to TR was seen in 10% of patients.

Signs of right heart failure

Raised JVP

Raised JVP was seen in 73 patients (i.e. 73 %) in our study secondary to RV failure.

Hepatomegaly

Hepatomegaly was present in 47 % of patients in our study secondary to congestive heart failure.

Pedal edema was seen in 77 % of our patient in our study.

Others

Four patients with ischemic dilated cardiomyopathy and AF had stroke which was secondary to cerebral embolism. Majority of the patients presented with biventricular failure.

Exertional dyspnea was found to be the most common symptom in our study being present in all our patients followed by symptoms like easy fatigability, pedal edema, cough, palpitation and abdominal pain. This presentation is similar to the clinical profile seen in many other studies.

In our study up to 35 % of patients had chest pain. This was high compared to other studies like S. Ahmad et alwhere in chest pain was seen in 29%. This could be due to inclusion of patients with ischemic cardiomyopathy as compared to the other study where it was excluded.

In addition syncope was present in up to 17 % of our patients in this study, whereas in other studies syncope was seen only in 1.8% (S. Ahmed et al) [60].This high number could again be attributed due to the inclusion of ischemic cardiomyopathy in our study. Arrhythmias and severe left ventricular dysfunction are more commonly present in ischemic cardiomyopathy may lead to syncope.

Physical findings	Our Study (%)	S. Ahmad et al (%)[60]
Basal crepitations	93	90
Pedal edema	77	67.2
Raised JVP	73	83
Hepatomegaly	47	54
LVS3	47	45
RVS3	20	-

Radiological features

Chest radiograph was found to be abnormal in almost all the cases showing varying degree of cardiomegaly with CT ratio varying between 0.5 to 0.75. This was similar to the study done by Masssumi et al, in that cardiomegaly was found in all cases with CT ratio between 0.51 to 0.80.

27 % of patients in our study had pleural effusion compared to 46% in the Massumi et al study and 10% in Ahmad et al study.

Pulmonary plethora was found in 51% as compared to 72% in Massumi et al study and 76.3% in the Ahmad et al study.

Parameter	Present study (%)	Massumi et al(%)[62]	Ahmad et al(%)[60]
Cardiomegaly	100	100	96.3
Pleural effusion	27	46	10
Pulmonary plethora	51	72	76.2

Electrocardiographic profile

The QRS axis was normal in 74 % of our subjects with left axis deviation in 17 % and right axis deviation in 9% which were in concordance with all the other studies. Sinus tachycardia was the most predominant finding in the S. Ahmad et al study being found in up to 69% of patients. Our study showed sinus tachycardia in 45 % of patients.

Other ECG parameters like ventricular ectopics, LBBB, Atrial fibrillation, atrial ectopics were comparable to those in all the other studies. However RBBB, complete heart block and SVT were more commonly present in our study as compared to other studies. These could again be due to the inclusion of ischemic cardiomyopathy in our study.
LVH was less commonly seen in our study being present in 22 % as compared to 30 to 40% in other studies.

Non specific ST-T changes were seen in 29% of cases in our study, similar to that in other studies.

Parameters		Our study	S. Ahmad et al[60]
QRS axis	Normal	74	70.9
	LAD	17	20
	RAD	9	9
LVH		22	40
ST-T changes		29	10
Arrhythmias	Ventricular ectopics	46	29.1
	Sinus tachycardia	45	69.1
	LBBB	42	32
	RBBB	13	-
	AF	14	9.1
	Atrial ectopics	11	5.4
	SVT	7	0
	VT	4	0
	CHB	3	10.9

Echocardiographic profile

The mean left ventricular EF in our study group was 30.34 %. This was similar to that in all the other studies on dilated cardiomyopathy. The mean LVEDD was 5.78 cm. The mean LVESD was 4.62 cm.

Mitral regurgitation was seen in 68 % of our patients in our study comparable to that in other study groups. Mitral regurgitation (68 %) was much more commonly seen when compared to tricuspid regurgitation (8 %). This was mainly due to large proportion of patients with ischemic DCM and severe LV dysfunction compared to nonischemicDCM. Six of our patients had AR in our study when compared to 17.8% of patients in Jain et al study. Mitral and tricuspid regurgitation in dilated cardiomyopathy are secondary to annular ring dilatation

Left ventricular clot was seen in four patients in our study who also had cerebral embolism secondary to atrial fibrillation. Pericardial effusion was seen in 9 % of our patients.

Parameter (mean)	Our study	Ahmad et al [60]	Jain et al [61]	G. Singh et al [63]	Routray et al [64]	Rihal et al [65]
LV EF	30.34	30.05	29	30	35	23
LV EDD	5.78	6.45	6.64	6.48	61	6.9
LV ESD	4.62	5.8	5.74	5.63	52.3	6
MR	68	63.6	67	90	86	43.13
TR	8	26.3	46	48	36	24
LV clot	4	3.6	4	6	11	6
Pericardial effusion	9	4	10.44	12.4	5.5	8.2

Etiological profile

In our study the most common type of dilated cardiomyopathy was ischemic dilated cardiomyopathy being present in 47 % of our patients, followed by alcoholic cardiomyopathy seen in 15 %. Diabetic cardiomyopathy wasfound to be the third most common type seen in 11 % of patients while Idiopathic and Peripartum cardiomyopathy were seen in 12 % and 9 % respectively. The miscellaneous group included 6 patients; one with post viral myocarditis, two patients had HIV cardiomyopathy and three had valvular cardiomyopathy. Ischemic cardiomyopathy was not included in most studies on dilated

cardiomyopathy due to the controversy in defining the term “Ischemic cardiomyopathy”.

In Jain et al study ischemic cardiomyopathy comprised 37% of cases followed by idiopathic dilated cardiomyopathy seen in 30% of patients. The incidence of idiopathic DCM in their study was much higher compared to our study. Other sub groups of DCM were comparable to our study.

Coronary angiography was done in all the patients with ischemic cardiomyopathy in our study. Of the 47 patients studied 29 of them had history of previous myocardial infarction. All the 47 patients in our study had significant narrowing of epicardial coronaries (i.e. > 70% of lumen). Twenty one patients had double vessel disease, six showed triple vessel disease and two had single vessel disease. The echocardiography of all the patients showed global hypokinesia with reduced ejection fraction.

Among the patients with alcoholic cardiomyopathy three had hepatic cirrhosis. Liver function tests revealed mildly raised bilirubin (1.5 mg %) with normal liver enzymes. This is similar to that found in other studies on alcoholic cardiomyopathy.

In our study 37 % of patients had anemia, most of the patients had mild anemia (i.e. Hb between 8.5 – 11 gm %). In a study done by A. Justin et al anemia was found in 27% of patients with congestive heart failure. The prevalence of anemia in our study is similar. Anemia is

known to be associated with adverse outcome in patients with heart failure.

Etiology	Our study (%)	Jain et al (%) [61]
Ischemic	47	37
Alcoholic	15	14.5
Idiopathic	12	30
Diabetic	11	7.8
Peripartum	9	7
Miscellaneous	6	3.7

Among alcoholics alcohol plays a significant etiological role (p value <0.0001)

The mean EF in our study was 31.2±7.5

The mean LVEDD in our study was 6.04±0.74

The mean LVESD in our study was 4.92±0.62

There is no significant difference between EF & LVEDD among ischemic and non ischemic DCM (p value 0.85 and p value 0.72 respectively)

SUMMARY

- The objective of the study was to know the etiology and clinical profile including ECG and ECHO changes in dilated cardiomyopathy 100 cases of dilated cardiomyopathy, of which 57 were males and 43 were female were included in the study.
- Dilated cardiomyopathy is common in the elderly and middle aged population and the etiology varies with age.
- Dilated cardiomyopathy is more common in males.
- Biventricular failure was the most common clinical presentation (77 %) followed by left heart failure (19 %) and then right heart failure (4%).
- The most common type was ischemic variety followed by alcoholic, idiopathic, diabetic and peripartum cardiomyopathy.
- The electrocardiographic profile showed ventricular ectopics, sinus tachycardia, left bundle branch block, atrial fibrillation, right bundle branch block, atrial ectopics, SVT, ventricular tachycardia and complete heart block.LVH was present in 22 % of cases.
- Chest radiography showed cardiomegaly in all the cases. Pulmonary plethora was noticed in significant number of patients (51 %). Pleural effusion was noticed in (27 %).
- Echocardiographic profile included reduced EF and global hypokinesia in all the patients. There was also varying degree of

left ventricular dilatation. Mitral regurgitation was seen in significant number of patients (68 %). Pericardial effusion was seen in 9 % of the patients.

- Most of the patients were in NYHA class IV (47 %) and class III (33%).
- Among alcoholics alcohol plays a significant etiological role (p value <0.0001)
- The mean EF in our study was 31.2±7.5
- The mean LVEDD in our study was 6.04±0.74
- The mean LVESD in our study was 4.92±0.62
- There is no significant difference between EF & LVEDD among ischemic and non ischemic DCM (p value 0.85 and p value 0.72 respectively)

CONCLUSION

The major cause of dilated cardiomyopathy in our study was found to be ischemic followed by alcoholic and idiopathic cardiomyopathy. The most common clinical presentation is biventricular failure. Chest radiography showed cardiomegaly in most patients. The common abnormality in ECG consists of sinus tachycardia, AF, LBBB. ECHO showed reduced EF and global hypokinesia universally. Mitral regurgitation and pericardial effusion were present in significant number of patients. Most of the patients presented with NYHA class 1V.

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KEY TO PROFORMA

PND-Paroxysmal nocturnal dyspnoea

IHD- Ischemic heart disease

MI-Myocardial infarction

DM-Diabetes mellitus

SHT-Systemic hypertension

BP-Blood pressure

JVP-Jugular venous pressure

CNS-Central nervous system

NYHA-Newyork heart association

ECG-Electrocardiogram

TC-Total count

DC-Differential count

ESR-Erythrocyte sedimentation rate

RFT-Renal function test

LFT-Liver function test

CT Ratio-Cardio thoracic ratio

EF-Ejection fraction

LVEDD-Left ventricular end diastolic dimension

LVESD-Left ventricular end systolic dimension

LA-Left atrium

RA-Right atrium

LV-Left ventricle

RV-Right ventricle

KEY TO MASTER CHART

DM-Diabetes mellitus

SHT-Systemic hypertension

IHD-Ischemic heart disease

PND-Paroxysmal nocturnal dyspnoea

JVP-Jugular venous pressure

LVS3-Left ventricular S3

RVS3-Right ventricular S3

PSM-Pan systolic murmer

NYHA-Newyork heart association

EF-Ejection fraction

LVEDD-Left ventricular end diastolic dimension

LVESD-Left ventricular end systolic dimension

B-Bradycardia

ST-Sinus tachycardia

AF-Atrial fibrillation

VPC-Ventricular premature contraction

CHB-Complete heart block

PROFORMA

Name: Age: Sex:
Address:
Unit/Ward:

PRESENT HISTORY

Breathlessness
Syncope
Orthopnoea
Palpitations
PND
Abdominal pain
Chest pain
Pedal edema
Cough

PAST HISTORY

IHD
Diabetes mellitus
Hypertension
Alcohol
HIV infection

PERSONAL HISTORY

Diet
Smoking
Alcohol
Tobacco

FAMILY HISTORY

DM
SHT
Premature IHD(<40yrs)

TREATMENT HISTORY

GENERAL PHYSICAL EXAMINATION

Weight
Anaemia
Jaundice
Cyanosis
Pedal edema
Lymphadenopathy
Pulse – Rate
Rhythm
Volume
Character
Other peripheral pulses:

	Right	left
Carotids		
Brachial		
Radial		
Femoral		
Popliteal		
Posterior tibial		
Dorsalispedis		

BP
Respiratory rate
JVP

EXAMINATION OF CARDIOVASCULAR SYSTEM:

INSPECTION – Apical impulse location
Pulsation other than apical impulse
Epigastric
Lt parasternal
Pulmonary area
Suprasternal
Supraclavicular

PALPATION- Apical impulse- location/character
Lt para sternal heave
Thrill
Palpable P2

Pulsations

PERCUSSION- Rt border
Lt border
Lt 2nd space

AUSCULTATION- Heart sounds S1,S2,S3
Murmer – Site
Character
Grade
Radiation

OTHER SYSTEMS

RESPIRATORY SYSTEM
ABDOMEN
CNS

DIAGNOSIS

NYHA Class

INVESTIGATIONS :

COMPLETE HEMOGRAM

Hb (g/dL)
TC (cells/cmm)
DC
RBC(millions/cmm)
Platelet(Lakhs/cmm)
ESR

RFT

Blood Sugar(mg/dL)
Blood Urea(mg/dL)
S.Creatinine(mg/dL)
S.Sodium(meq/l)
S.Potasium(meq/l)

LFT

S.Bilirubin(mg/dL) T:
D:
Protein
SGOT(IU/L)
SGPT(IU/L)
SAP(IU/L)

Chest X ray – CT ratio
Pulmonary vasculature
Pleural effusion

ECG- Rate
Rhythm
Axis
Pwave
QRS
Twave
STsegment
Conclusion

ECHOCARDIOGRAM

Measurement:
EF -
LVEDD -
LVESD -

Chambers:
LA-
RA-
LV-
RV-

Doppler study – Mitral valve
Tricuspid valve
Aortic valve
Pulmonary valve

PE
Thrombus/vegetation

Others:

MASTER CHART

				HISTORY			SYMPTOMATOLOGY									PHYSICAL FINDINGS									ECHO			
S.No	NAME	AGE	SEX	DM	IHD	ALCOHOL INTAKE	DYSPNOEA (GRADE)	PND	ORTHOPNOEA	PALPITATION	CHEST PAIN	COUGH	ABDOMINAL PAIN	EASY FATIGUABILITY	SYNCOPE	PEDAL EDEMA	PULSE	BASAL CREPITATIONS	RAISED JVP	HEPATOMEGALY	LV S3	RV S3	PSM	SBP<100 mmHG	NYHA	EF	LVEDD	LVESD	ETIOLOGY
1	KANNAN	47	M	-	+	+	3	+	+	+	+	+	-	+	-	-	AF	+	+	-	+	-	-	-	3	35	5.4	4.6	ISCHEMIC
2	RAMASAMY	60	M	+	+	+	4	-	-	+	+	+	+	+	-	+	ST	+	+	+	-	-	+	+	4	19	6.6	5.4	ISCHEMIC
3	MURUGAN	61	M	-	-	+	4	+	+	+	-	-	-	+	-	-	ST	+	-	-	-	-	-	+	4	21	6.8	5.6	ALCOHOLIC
4	AMUDHA	36	F	-	-	-	3	-	-	-	-	+	-	+	-	+	AF	-	-	+	-	+	+	-	3	36	6.4	5.5	PERIPARTUM
5	RAVI	65	M	-	-	+	2	-	-	+	-	-	-	-	-	-	ST	+	+	-	-	-	-	-	2	40	6.5	5.2	ALCOHOLIC
6	SHANTHI	19	F	-	-	-	4	+	+	-	-	+	+	+	-	+	B	+	+	+	+	-	+	+	4	23	6.9	5.5	IDIOPATHIC
7	PONNUSAMY	51	M	+	-	-	4	-	-	-	-	-	-	+	+	+	ST	+	-	-	-	-	+	+	4	34	6.6	5.2	DIABETIC
8	MANOHAR	72	M	-	-	+	3	+	+	+	-	+	-	+	-	-	ST	+	+	+	-	-	-	-	3	23	5.4	4.5	ALCOHOLIC
9	SAMPATH	66	M	-	+	+	2	-	-	+	+	+	+	-	-	+	VPC	+	+	-	-	-	+	-	2	43	4.5	3.9	ISCHEMIC
10	SIVASHANKAR	48	M	-	+	-	3	+	+	-	+	+	-	+	+	+	ST	+	-	+	+	-	-	-	3	37	5.4	4.2	ISCHEMIC
11	ELANGO	61	M	-	-	+	4	+	+	+	-	+	-	+	-	+	ST	-	+	+	+	-	+	+	4	18	6.8	5.6	ALCOHOLIC
12	SAROJA	41	F	+	+	-	4	+	+	-	-	-	+	+	-	-	B	+	+	-	-	-	-	-	4	23	6.4	5.5	ISCHEMIC
13	MARIAMMAL	46	F	+	-	-	3	-	-	+	-	+	+	+	-	+	ST	+	+	+	-	+	+	-	3	35	5.4	4.9	DIABETIC
14	DEVA	58	M	-	-	-	3	+	-	+	-	+	-	+	-	+	VPC	+	+	-	+	-	+	-	3	34	6.4	5.2	IDIOPATHIC
15	KRISHNAMURTHY	72	M	-	+	+	4	-	-	+	+	+	-	+	-	-	AF	+	-	+	-	-	-	+	4	24	6.6	5.6	ISCHEMIC
16	KUMARAN	68	M	-	+	+	2	-	-	-	+	-	-	-	-	+	ST	-	-	+	-	-	+	-	2	42	4.6	3.6	ISCHEMIC
17	TAMIZHAN	67	M	-	+	+	2	+	-	+	+	+	-	+	+	+	ST	-	-	-	+	-	+	-	2	41	4.6	3.9	ISCHEMIC
18	JEYANTHI	61	F	+	+	-	4	+	+	+	+	+	+	+	-	+	ST	+	+	-	+	-	-	+	4	25	6.8	5.5	ISCHEMIC
19	THENMOZHI	62	F	-	+	-	4	+	+	+	+	+	-	+	-	-	AF	+	+	-	-	-	+	+	4	18	6.4	5.3	ISCHEMIC
20	SANTHANAM	66	M	-	-	-	3	+	-	+	+	+	+	+	-	+	VPC	+	-	+	-	+	+	-	3	34	5.4	4.6	IDIOPATHIC
21	SUNDARAM	46	M	-	-	-	3	+	+	+	-	-	-	-	-	+	ST	+	+	+	-	+	-	-	3	43	5.4	4.9	HIV
22	MANIGANDAN	65	M	-	+	+	4	+	+	+	+	-	+	-	-	+	VPC	-	+	-	-	+	+	+	4	26	6.9	5.5	ISCHEMIC
23	GURUSAMY	52	M	-	-	+	3	+	-	+	-	+	+	+	-	-	VPC	+	+	+	-	-	-	-	3	38	5.3	4.6	ALCOHOLIC
24	RANI	59	F	+	-	-	2	-	-	+	-	-	-	-	+	+	ST	+	+	+	-	+	-	+	2	42	4.5	3.5	DIABETIC
25	MALAYAPPAN	64	M	-	+	-	2	-	-	+	+	+	+	+	-	-	VPC	+	-	+	-	+	-	-	2	44	4.5	3.6	ISCHEMIC

MASTER CHART

				HISTORY			SYMPTOMATOLOGY									PHYSICAL FINDINGS									ECHO			
S.No	NAME	AGE	SEX	DM	IHD	ALCOHOL INTAKE	DYSPNOEA (GRADE)	PND	ORTHOPNOEA	PALPITATION	CHEST PAIN	COUGH	ABDOMINAL PAIN	EASY FATIGUABILITY	SYNCOPE	PEDAL EDEMA	PULSE	BASAL CREPITATIONS	RAISED JVP	HEPATOMEGALY	LV S3	RV S3	PSM	SBP<100 mmHG	NYHA	EF	LVEDD	LVESD	ETIOLOGY
26	RAMANATHAN	51	M	+	+	-	4	+	+	+	-	+	+	+	-	+	ST	+	+	+	-	+	-	+	4	28	6.6	5.2	ISCHEMIC
27	RANGANATHAN	60	M	+	-	-	4	+	+	-	-	+	-	+	+	+	ST	+	-	+	-	-	-	+	4	28	6.8	5.3	DIABETIC
28	PORKODI	71	F	-	+	-	3	+	-	+	+	+	-	+	-	+	VPC	+	+	-	+	-	+	-	3	38	5.3	4.2	ISCHEMIC
29	RAMALAKSHMI	51	F	+	+	-	3	-	-	-	-	+	-	+	-	-	ST	+	+	+	+	-	+	-	3	38	6.4	5.4	ISCHEMIC
30	ELAVARASAN	56	M	-	-	+	4	+	+	+	-	+	+	+	-	+	AF	+	-	-	+	-	-	-	4	28	6.9	5.5	ALCOHOLIC
31	PANDIYAN	63	M	-	-	-	3	-	-	-	-	-	-	+	+	+	ST	+	+	+	-	+	-	-	3	36	5.3	4.9	IDIOPATHIC
32	GANDHIMATHI	38	F	-	-	-	4	+	+	+	-	+	-	+	-	+	ST	+	-	+	+	-	+	+	4	26	6.6	5.5	PERIPARTUM
33	MALARKODI	56	F	-	+	-	3	-	-	-	+	-	+	+	-	-	VPC	+	+	+	+	-	-	-	3	21	6.8	5.3	ISCHEMIC
34	SATHYAMURTI	60	M	-	+	-	2	-	-	+	+	+	-	-	-	+	ST	-	-	-	-	-	-	-	2	41	4.6	3.9	ISCHEMIC
35	PUSHPARANI	47	F	-	-	-	2	-	-	-	-	+	-	-	-	+	ST	+	+	+	-	-	+	-	2	43	5.5	4.9	IDIOPATHIC
36	KUPPUSAMY	44	M	-	-	-	4	+	+	-	-	-	+	+	+	+	AF	+	-	+	+	-	-	+	4	19	5.5	4.6	HIV
37	PREMAKUMARI	69	F	-	+	-	4	+	+	+	+	+	+	+	-	-	B	+	+	+	-	-	-	-	4	23	6.7	5.6	ISCHEMIC
38	KODANDARAMAN	36	M	+	+	-	3	-	-	+	+	-	+	+	-	+	ST	+	+	-	+	-	+	-	3	23	5.3	4.5	ISCHEMIC
39	VIJAYALAKSHMI	67	F	-	+	-	3	-	-	+	-	+	-	+	+	+	VPC	+	+	+	+	-	+	-	3	23	6.9	5.6	ISCHEMIC
40	PURUSHOTHAMAN	61	M	-	-	-	2	-	-	+	-	-	-	+	-	-	ST	+	+	-	+	-	-	-	2	42	4.7	3.5	IDIOPATHIC
41	GURUMURTI	55	M	-	-	+	3	-	-	+	-	+	+	+	-	-	ST	+	+	+	-	+	-	-	3	38	5.3	4.2	ALCOHOLIC
42	SRINIVASAN	45	M	-	+	+	3	-	-	+	-	-	-	+	-	-	ST	+	+	-	+	-	-	-	3	36	5.5	4.6	ISCHEMIC
43	VIJAYALAKSHMI	62	F	+	+	-	3	+	+	-	+	+	-	-	+	+	AF	+	+	-	+	-	+	-	3	38	5.5	4.9	ISCHEMIC
44	VASANTHI	65	F	+	+	-	4	+	+	+	+	-	+	+	-	+	ST	+	-	+	-	+	+	+	4	24	6.6	5.4	ISCHEMIC
45	RAMANATHAN	56	M	+	-	+	4	+	+	+	+	+	-	+	-	+	VPC	+	+	-	+	-	-	+	4	25	6.8	5.2	DIABETIC
46	RAJAKUMARAN	35	M	-	-	+	4	+	+	-	-	-	+	+	+	-	ST	+	+	+	+	-	+	+	4	18	6.4	5.3	ALCOHOLIC
47	MARIAMMAL	55	F	-	-	-	3	-	-	+	-	+	+	+	-	+	ST	+	-	-	+	-	-	-	3	38	5.5	4.2	IDIOPATHIC
48	RAMAMURTHY	66	M	-	+	+	1	-	-	+	+	-	-	-	-	-	VPC	+	+	+	-	+	+	-	1	44	4.8	3.9	ISCHEMIC
49	VENNILA	29	F	-	-	-	3	+	+	-	-	+	-	+	-	+	VPC	+	+	-	-	-	-	-	3	38	5.5	4.6	PERIPARTUM
50	VENUGOPAL	38	M	+	+	-	2	-	-	+	-	+	-	+	-	+	ST	+	-	+	+	-	+	-	2	41	6.9	5.5	ISCHEMIC

MASTER CHART

				HISTORY			SYMPTOMATOLOGY								PHYSICAL FINDINGS										ECHO			
S.No	NAME	AGE	SEX	DM	IHD	ALCOHOL INTAKE	DYSPNOEA (GRADE)	PND	ORTHOPNOEA	PALPITATION	CHEST PAIN	COUGH	ABDOMINAL PAIN	EASY FATIGUABILITY	SYNCOPE	PEDAL EDEMA	PULSE	BASAL CREPITATIONS	RAISED JVP	HEPATOMEGALY	LV S3	RV S3	PSM	SBP<100 mmHG	NYHA	EF	LVEDD	LVESD	ETIOLOGY
51	PRABHAKARAN	50	M	-	+	+	2	-	-	+	+	-	+	+	-	-	ST	+	+	-	+	-	+	-	2	40	5.5	4.5	ISCHEMIC
52	PRABHAKARAN	61	M	-	+	-	4	+	+	-	+	+	-	+	+	+	VPC	+	+	+	+	-	+	+	4	26	6.6	5.2	ISCHEMIC
53	ARIVALAGAN	51	M	+	-	+	1	-	-	+	--	+	-	-	-	+	ST	+	+	-	-	-	-	-	1	42	4.9	3.6	ALCOHOLIC
54	KOWSALYA	60	F	-	-	-	4	+	+	+	-	-	+	+	-	-	VPC	+	+	+	-	+	-	+	4	26	6.8	5.5	VALVULAR
55	MADURAIVELAN	59	M	-	-	+	4	+	+	-	-	+	-	+	-	+	VPC	+	+	-	+	-	-	+	4	28	6.4	5.6	ALCOHOLIC
56	NIRMALA	27	F	-	-	-	4	+	+	-	-	+	-	+	-	-	VPC	+	+	+	-	-	-	-	4	28	5.9	4.5	PERIPARTUM
57	PANNERSELVAM	67	M	-	-	-	3	+	+	-	-	-	+	+	-	-	ST	+	+	+	+	-	+	-	3	38	5.5	4.9	IDIOPATHIC
58	VIJAYALAKSHMI	62	F	+	+	-	4	+	+	-	+	+	-	+	-	+	AF	+	-	-	+	-	+	+	4	34	6.9	5.4	ISCHEMIC
59	KRISHNAN	68	M	+	-	+	3	+	-	+	-	-	-	+	+	+	ST	+	+	+	-	+	-	-	3	38	5.7	4.5	DIABETIC
60	RANIAMMAL	65	F	-	+	-	4	+	+	-	+	+	-	+	-	+	ST	+	+	-	+	-	+	+	4	26	6.6	5.2	ISCHEMIC
61	KUMARAN	39	M	-	-	-	3	-	-	+	-	-	+	+	-	+	VPC	+	-	-	+	-	-	-	3	34	5.7	4.6	IDIOPATHIC
62	PAVALAVENI	60	F	+	+	-	4	+	+	+	+	+	-	+	-	-	AF	+	+	+	-	-	-	+	4	21	6.8	5.2	ISCHEMIC
63	MUTHUKUMARI	61	F	-	+	-	4	+	+	-	+	-	-	+	+	+	ST	+	-	+	-	+	-	+	4	19	6.4	5.3	ISCHEMIC
64	GURUBARAN	69	M	-	-	+	3	-	-	+	-	+	+	+	-	+	ST	+	+	-	-	-	+	-	3	23	5.7	4.2	ALCOHOLIC
65	MANIMEGALAI	72	F	-	+	-	3	+	+	-	-	+	-	+	-	-	ST	-	+	+	+	-	+	-	3	23	5.7	4.7	ISCHEMIC
66	MANIKUMAR	70	M	+	-	-	2	-	-	+	-	-	-	-	-	+	ST	+	-	-	+	-	-	-	2	31	4.7	3.9	DIABETIC
67	MURUGESAN	39	M	-	+	+	2	-	-	+	-	+	+	-	-	-	ST	+	+	+	-	+	+	-	2	43	6.7	5.5	ISCHEMIC
68	MALATHY	56	F	-	-	-	2	-	-	+	-	-	-	+	-	+	AF	+	+	-	-	-	-	-	2	31	5.8	4.6	VALVULAR
69	RAJADEVI	60	F	+	+	-	4	+	+	-	+	+	-	+	-	-	ST	+	+	+	+	-	+	+	4	24	6.6	5.3	ISCHEMIC
70	MURUGANATHAN	71	M	-	+	+	4	+	+	-	+	-	+	+	-	+	VPC	+	+	-	+	-	-	-	4	25	6.8	5.2	ISCHEMIC
71	RAMASUBBU	35	M	-	+	+	4	+	+	-	-	+	+	+	-	+	ST	+	-	+	-	+	+	+	4	26	6.4	5.4	ISCHEMIC
72	KANDASAMY	63	M	-	-	+	2	-	-	+	-	-	-	+	-	+	B	+	+	+	+	-	-	-	2	38	4.8	3.5	ALCOHOLIC
73	SUSEELA	36	F	-	-	-	3	+	+	-	+	+	-	-	-	-	ST	+	+	-	+	-	+	-	3	38	5.7	4.9	PERIPARTUM
74	RAMARPILLAI	64	M	-	-	+	4	+	+	-	-	-	-	+	-	+	ST	+	+	+	-	+	+	+	4	28	6.9	5.6	ALCOHOLIC
75	RUPAVATHI	59	F	-	-	-	3	-	-	+	-	+	-	+	-	+	ST	-	-	-	+	-	-	-	3	36	5.9	4.5	IDIOPATHIC

MASTER CHART

				HISTORY			SYMPTOMATOLOGY									PHYSICAL FINDINGS									ECHO			
S.No	NAME	AGE	SEX	DM	IHD	ALCOHOL INTAKE	DYSPNOEA (GRADE)	PND	ORTHOPNOEA	PALPITATION	CHEST PAIN	COUGH	ABDOMINAL PAIN	EASY FATIGUABILITY	SYNCOPE	PEDAL EDEMA	PULSE	BASAL CREPITATIONS	RAISED JVP	HEPATOMEGALY	LV S3	RV S3	PSM	SBP<100 mmHG	NYHA	EF	LVEDD	LVESD	ETIOLOGY
76	MUTHUSAMY	65	M	+	-	+	4	+	+	+	-	-	+	+	-	-	VPC	+	+	+	-	+	+	+	4	28	6.6	5.2	DIABETIC
77	KAMALA	71	F	-	+	-	3	+	-	+	-	+	-	+	+	+	ST	+	+	-	-	-	-	-	3	38	6.8	5.4	ISCHEMIC
78	SELVI	29	F	-	-	-	4	+	+	-	-	-	-	+	+	+	ST	+	-	-	+	-	+	-	4	27	6.4	5.2	PERIPARTUM
79	THAMBIDURAI	66	M	-	+	+	2	-	-	+	+	+	-	-	-	+	ST	+	+	-	+	-	+	-	2	39	6.9	5.3	ISCHEMIC
80	PADMAVATHY	70	F	+	+	-	3	-	-	+	+	+	-	+	-	-	AF	+	+	-	+	-	-	-	3	36	5.7	4.9	ISCHEMIC
81	MADIALAGAN	67	M	-	+	+	4	+	+	+	-	+	+	+	-	+	ST	+	+	+	-	-	+	-	4	26	6.6	5.5	ISCHEMIC
82	POORNIMA	34	F	-	-	-	2	-	-	+	-	-	-	+	-	+	AF	+	+	-	+	-	+	-	2	44	4.9	3.6	PERIPARTUM
83	MURUGAN	68	M	-	+	+	3	-	-	+	+	+	-	+	-	-	ST	+	-	-	-	-	-	-	3	34	5.9	4.2	ISCHEMIC
84	KALAISELVI	61	F	+	+	-	4	+	+	-	-	+	-	+	-	+	VPC	+	+	+	+	-	+	+	4	21	6.8	5.6	ISCHEMIC
85	KUPPUSAMY	71	M	-	+	+	4	+	+	-	-	-	+	+	-	+	ST	+	+	-	-	-	-	-	4	23	5.8	4.9	ISCHEMIC
86	PANDIDURAI	38	M	-	-	+	4	+	+	-	-	+	-	+	-	+	ST	+	+	-	+	-	+	+	4	23	6.4	5.3	ALCOHOLIC
87	PUSHPAVATHY	58	F	-	-	-	1	-	-	+	-	+	-	-	-	+	VPC	+	+	+	+	-	-	-	1	38	4.9	3.9	IDIOPATHIC
88	KANDASAMY	63	M	+	-	-	4	+	+	-	-	+	+	+	-	+	ST	+	+	-	-	-	+	-	4	24	5.8	4.5	DIABETIC
89	AMBIKA	60	F	-	+	-	4	+	+	-	-	+	-	+	-	+	AF	+	+	-	-	-	-	-	4	25	6.9	5.6	ISCHEMIC
90	KAMALA	64	F	-	+	-	3	-	-	+	+	-	-	+	-	+	ST	+	+	-	+	-	+	-	3	36	5.8	4.9	ISCHEMIC
91	KAMALAMMAL	62	F	+	+	-	4	+	+	-	-	+	-	+	-	+	ST	+	+	-	+	-	+	-	4	26	6.6	5.6	ISCHEMIC
92	DHANDAPANI	65	M	-	-	+	4	+	+	+	-	-	-	+	-	+	VPC	+	+	-	-	-	-	-	4	38	6.8	5.6	ALCOHOLIC
93	SURIYAPRIYA	55	F	-	-	-	4	+	+	-	-	-	+	+	-	+	B	+	-	-	+	-	+	-	4	28	6.9	5.5	IDIOPATHIC
94	RAMANATHAN	66	M	+	-	-	3	-	-	+	-	+	-	+	-	-	ST	-	+	+	-	+	-	-	3	31	5.9	4.6	DIABETIC
95	PRIYAKUMARI	51	F	-	-	-	4	+	+	-	-	-	-	+	+	+	ST	+	+	-	-	-	+	+	4	28	6.9	5.6	VALVULAR
96	ASHOKKUMAR	18	M	-	-	-	4	+	+	-	-	-	-	+	-	+	VPC	+	+	-	-	-	-	-	4	28	6.6	5.5	POST VIRAL MYOCARDITIS
97	BEEVIJOHN	36	F	-	-	-	4	+	+	+	-	+	-	+	-	+	ST	+	-	-	+	-	-	-	4	32	6.8	5.6	PERIPARTUM
98	ELANKUMARAN	67	M	+	-	+	4	+	+	-	-	-	-	+	-	+	B	+	+	-	-	-	-	-	4	32	6.4	5.3	DIABETIC
99	MARIAMMAL	38	F	-	-	-	3	-	-	+	-	-	-	-	+	+	ST	+	+	-	-	-	-	-	3	34	5.8	4.9	PERIPARTUM
100	FATHIMA	60	F	-	+	-	4	+	+	-	+	-	-	+	-	+	AF	+	+	-	-		-	-	4	26	6.9	5.6	ISCHEMIC

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. C.R. Srinivasan
PG in MD General Medicine
Madras Medical College, Chennai -3.

Dear Dr. C.R Srinivasan

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled " A study on etiology and clinical profile of dilated cardiomyopathy in Rajiv Gandhi Govt. General Hospital, chennnai" No. 03042011.

The following members of Ethics Committee were present in the meeting held on 21.04.2011 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan, MD	-- Chairperson
2.	Prof. V. Kanagasabai MD Dean, Madras Medical College, Chennai-3,	-- Deputy chairman
3.	Prof. A. Sundaram, MD Vice Principal , Madras Medical College, Chennai -3	-- Member Secretary
4.	Prof R. Sathianathan MD	-- Member
5.	Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	-- Member
6.	Prof. Pregna B. Dolia MD Director , Institute of Biochemistry, MMC, Ch-3	-- Member
7.	Prof. C. Rajendiran .MD Director , Institute of Internal Medicine, MMC, Ch-3	-- Member
8.	Thiru. A. Ulaganathan Administrative Officer, MMC, Chennai -3	-- Layperson
9.	Thiru. S. Govindasamy . BA.BL	-- Lawyer
10.	Tmt. Arnold Soulina	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee